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The Journal of Neurology and Psychopathology

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THE JOURNAL OF NEUROLOGY AND PSYCHOPATHOLOGY.

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Original Papers.

THE PROBLEM OF JUVENILE BEHAVIOUR DISORDERS IN CHRONIC EPIDEMIC ENCEPHALITIS.*

By T. R. HILL, London.

The manifestations of chronic epidemic encephalitis are so varied and, in individual cases, so liable to change in nature with the passage of time that it is almost impossible to put cases satisfactorily in a permanent classification. Nevertheless, cases can be classified at any one time into broad groups, each with a prominent and characteristic feature. The largest of these is undoubtedly the Parkinsonian group, but one of the most outstanding is that of behaviour disorders in children. In contrast to the knowledge only too readily gained of the clinical picture presented by these latter cases is the obscurity of the morbid processes producing the disorders. It is the problem of the precise nature of these morbid processes with which it is proposed to deal in this communication. The incidence of the psychological changes in general and the behaviour disorders in particular in children attacked by epidemic encephalitis will first be shown ; the clinical features of the behaviour disorders, with three illustrative cases, will then be described and, finally, theories of pathogenesis quoted and discussed.

INCIDENCE.

Grossman, of 89 cases, found over 50 per cent. with mental abnormalities, and Hall, commenting on these figures, said that slight changes were probably present in many more. Wimmer stated that of 25 children attacked 11 died, two were untraced, and the remaining 12 all showed pronounced psychological changes. Shrubsall, examining 119 children nine months after the acute attack, found 44 with conduct changes. Of 67 cases under our own observation 26 show behaviour disorders ; 23, pure Parkinsonism ; nine, a mixture of these two ; five, serious mental retardation (only one definitely feeble-minded) ; and five are mentally normal. These figures indicate that of children attacked by epidemic encephalitis over 70 per cent. of survivors show some psychological changes and more than a third show behaviour disorders.

* From the Post-Encephalitis Lethargica unit of the Metropolitan Asylums Board, Winchmore Hill.

CLINICAL FEATURES.

According to Wimmer, of all cases of behaviour disorder changes in morals and character stamp the picture, revealing curious uniformity in different patients. Psychical changes may occur at the outset of the disease, but in most cases they develop at a later period. The following is Wimmer's list of characteristic conduct changes: restlessness, garrulousness, meddlesomeness, wide-awake yet erratic attention, excessive curiosity, moods of foolish mirthfulness, irritability, queer manners, desire to tease, querulousness, marked emotional irritability with uncontrolled outbursts of anger, scolding and the use of abusive language, propensity to destructiveness, biting nails, auto-mutilations, smearing objects with fæces and urine, wilful spitting or micturition on surrounding persons or objects, violence, attempts at murder, attempts at setting fire to hospital beds, cruelty towards other children or animals, truancy from school, vagrancy, begging, dishonesty, mendacity, denunciations and pilferings; very commonly precocious eroticism, associated sometimes with premature puberty leading to masturbation, obscene language and conduct, exhibitionism, attempts on adult females or offences to small girls. The children, says Wimmer, become backward at school, perhaps primarily on account of their lack of mental perseverance. Any of the above disturbances may be present in a mixed type with a degree of Parkinsonism, though not very frequently. He adds that it is doubtful whether actual defects of intelligence are present in the majority of cases and that some may be of the 'nocturnal' type, i.e., the patients' behaviour is normal during the daytime, but becomes disordered at a fixed hour in the evening and continues so during the night. Any of the above conduct changes may be associated with signs of organic or visceral disease, e.g., tics, respiratory troubles, etc.

The above description covers exactly the picture presented by our 26 cases. The following points may be added.

Misbehaviour, whether immediately following the acute attack or appearing only after some months, develops to a maximum fairly rapidly, and then runs a steady course for years, not progressing or retrogressing, except when in some cases Parkinsonism supervenes and, when pronounced, abolishes it. The cases may continue in a mixed state but behaviour disorders do not occur in severe, and are usually lessened by mild, Parkinsonism. The children show at frequent intervals any of the forms of abnormal behaviour mentioned above. A severe case may be continually misbehaved, a mild case remain normal except for occasional outbursts. In all but the mildest cases the patients have in common a liability to bouts of anger and fighting, and a general restlessness with secondary inability to sit still for long or concentrate in school. Attempts at mental concentration make their restlessness and behaviour worse, whilst occupations not demanding much sustained concentration usually improve them for the time. However, they are fundamentally uninfluenced by environmental

changes, the effect of discipline, etc., being quite superficial. Very mild cases may appear normal under the latter, but relapse when the patients return home.

Many of our patients are physically normal, though, as mentioned above, nine show a mild degree of Parkinsonism. Ocular abnormalities, tics, and nocturnal insomnia are common. There is one case of moderate dystrophia adiposogenitalis but none of premature puberty.

The patients never suffer from a gross defect in intelligence. In nearly all the parents say with certainty that no falling off in general intelligence was apparent to them. Anderson states that the children "do not suffer serious mental defect and that conversely severe impairment is not accompanied by behaviour disorders in any marked degree," and she adds that a noteworthy characteristic is that all have excellent memories. Also, Marshall has found that the general intelligence is not appreciably affected and that it is quite exceptional to find an intelligence quotient below 85. A recent investigation by Dawson and Conn in Glasgow showed that all children attacked by encephalitis showed some small falling off in response to intelligence tests compared with controls. The mental ratio of 974 non-encephalitic patients averaged 90·53 and of 46 encephalitic patients 84·63. This not very greatly reduced response was common to all encephalitic children besides those with conduct changes. These investigators conclude as follows: "Whatever may be the ultimate explanation of the moral and emotional instability it does not appear to be due to intellectual degeneration." Of our patients a number of the most marked cases of behaviour disorder give a normal response to intelligence tests of their appropriate age and none shows serious retardation.

All the forms of misbehaviour are of a primitive and instinctive type of reaction and their immediate cause appears to be a slavery of the children to primitive and instinctive tendencies. These tendencies, which are present but much more under intelligent control in the normal child, seem to have their normal affective accompaniment so greatly exaggerated that obedience to them, in spite of the child's intelligence, is compulsory. The pleasure of satisfying them is so seductive and the pain of obstructing them so unbearable that sensual, crude and instinctive forms of behaviour have to be indulged in. There seems a general increase in the affective side of the child's life; and in a child whose behaviour is so much more than in an adult immediately determined by instinctive tendencies and responses, whose power of intelligent reflection and control is so undeveloped, and who is normally so ready to satisfy its appetites, to blaze into anger or dissolve into tears, it is easy to see how an increase in affective response to stimuli, whether mental or physical, pleasurable or painful, can exaggerate primitive and instinctive tendencies and produce their omnipotence.

The children realise the atrociousness of their actions, often showing spontaneous regret for them and evidence of trying to control themselves, weeping or giving signs of the most genuine repentance and complaining that

they "could not help it." Thus Anderson says: ". . . they appear to be acutely conscious of the impulse to do wrong, but are quite unable to control it." They frequently express their inability to hold themselves in check. A patient of Anderson's made this remark: "I am a bad boy. I know I am a bad boy, but I cannot help it." A patient of mine (Case 2) on sitting down at a table pushed a porcelain vessel out of her reach for fear she might have an uncontrollable impulse to smash it and, having spat fiercely at me, burst into tears and would not be consoled, blaming herself bitterly. Another patient said he often had a "feeling" that he must go into his garden and hurt his dog.

The great exaggeration in these patients of primitive and instinctive tendencies and their consequent slavery to them accounts for their general restlessness and inability to concentrate on intellectual work, although they often wish to and are distressed because they cannot (Cases 2 and 3), for they continually succumb from minute to minute to fresh impulses. Advice, admonition and punishment, though the patient receives them with genuine penitence and promises of amendment, are without effect, for primitive tendencies are naturally uninfluenced, and, as soon as these arise again, the patients relapse.

Tics have been mentioned above as occurring in cases of behaviour disorder, and occasionally (Case 3) some of the actual disorders of behaviour are of a tic-like nature, i.e., the same piece of misbehaviour of short duration is repeated in identical form over and over again for no apparent reason. Kinnier Wilson has emphasized the importance in the causation of tics of a seeking by the patients for a pleasurable sensation and of their susceptibility to "impressions heavily charged with pleasurable feeling-tone." This would explain their presence in these encephalitic children whom the disease has made so susceptible to states of pleasure or displeasure.

ILLUSTRATIVE CASES.

CASE 1. Female, age 15. Before the onset of encephalitis the patient was in every way quite normal, well-behaved, well-liked, fairly good at school and quite obedient. There was no neuropathic history. In February, 1922, at the age of nine years, she had a typical acute attack of epidemic encephalitis, with diplopia and lethargy which persisted for a month ("she slept 23 hours a day"). In the course of another month this slowly passed away and she seemed quite normal. For six months she continued quite well and then started quite suddenly quarrelling with other children at school or in the street. She grew very inattentive and developed a violent temper, which was extremely easily aroused and during which she would strike her mother fiercely. All this grew worse and worse until she became completely unmanageable at home. If any of her desires were thwarted, e.g., if she were not given her meals directly she wanted them, she would fly at her mother.

Since admission in 1926 she has shown considerable disorder of conduct. She is continually interfering with other children's occupations; she will issue orders to them, try to 'boss' them, and a fight will ensue. Her very violent, easily aroused temper produces fierce fights. When her interference with the other children is prevented by anyone, she will attack the latter, striking her and trying to seize her by the throat. Her interference with other children is governed by a curious maternal or protective

feeling for them, which leads her occasionally to order the nurses to carry out this or that treatment for them. Her life and actions in the hospital seem largely governed by this feeling. She fought with 'maniacal' temper one morning to get at a child from whose eyes she wished to remove a bandage. Her violent outbursts of temper, fighting, or defiance last only a few moments and are of daily occurrence. After them she cries with genuine repentance and expresses her sorrow and her inability to control herself. Her insight is very clear. She realises her uncontrollable desire to interfere with the other children, and after an outburst will say: "I am ashamed of myself. I went mad. I did not know what I was doing." However, in addition she will often make excuses and try to justify herself. She is often quite pleasant and normal for short periods, though minor delinquencies, e.g., thefts of food and lying to gain advantages, are very constant. The ward staff make this comment on her: "She seems to lose all control and then does not mind what she does to anybody, regardless of consequences." Punishment, reproof and appeal, though productive of tearful response and promises of good behaviour at the time, produce absolutely no effect in the long run. Since her admission, in spite of discipline and occupation, her behaviour disorder has shown no improvement. She has a very voracious appetite.

Physical development is normal. There is no sexual misbehaviour or premature puberty. There is moderate nocturnal restlessness, but no diurnal lethargy. Her intelligence appears unimpaired. In the words of her parents: "No loss whatever noticed in her intelligence, judgment or memory." In 1926 at the age of 13 her mental age was 12+, "but attention tires readily." Physically: pupils, right larger than left. Both sluggish to light and accommodation; marked coarse nystagmus in all directions; visual acuity normal. No extra-ocular paresis, but frequent transient diplopia. Otherwise normal; no trace of Parkinsonism or visceral involvement.

Case 2. Female, age 13. Before her illness the patient was a "sweet-tempered, bright and happy child." She had been very clever at school and there was no neuropathic history. In March, 1924, at the age of nine, she had an acute attack of epidemic encephalitis of sudden onset, with headache, fever, weakness of limbs, delirium and complete insomnia for three days and nights. Then for a fortnight she remained in bed, very drowsy, sleeping for "hours and hours." She then got up but continued to suffer from great nocturnal restlessness and diurnal lethargy. Five months later she seemed to have recovered and to be almost herself again. For one year she remained at school, never missing a day. Then she began to get very drowsy again during the day, "had no life," and did not want to go out at all. She slept all night and on rising would fall asleep over her breakfast, go to school and promptly fall asleep as soon as she got there, not being able to do her work. She developed at the same time frequent bouts of "deep-breathing" and had "queer turns," during which she would go quite rigid for a few moments. Tonsillectomy was performed in hospital. Immediately following the operation her condition became much worse. She started habits of spitting at and pinching other people, grinding her teeth, and used to cry a great deal (October, 1925). For the next four months, until admission to hospital, she was unmanageable at home, not only because of these impulsive actions, but because of other abnormal conduct, such as taking articles of her parents out of the house and giving them away.

After admission to hospital in February, 1926, the patient's behaviour and mental condition were reported as normal, except that she was apt to hang her tongue out frequently (a tic) and had bouts of noisy tachypnœa when excited. She ate and slept well and was good at school. Seven months after admission a change gradually appeared. She became constantly restless, more pert than usual to adults, was incessantly grinding her teeth when unoccupied, and developed numerous impulsive actions. She would suddenly tear her clothes, or smack someone in the face, or, if nobody were near, smack herself on the head, and she took to blowing at people who went near her and attempting

to spit at them, and was constantly spitting when walking about. She was quite conscious of her sins and ashamed and tearful on being spoken to about them, but said, "I can't help it," or "something makes me do it," and added that it "helped" her to smack someone and that it gave her the same satisfaction to smack her own head if no one else were present. She began to smash chinaware whenever she could, and to tear bed-clothes (making one sudden rip). She appeared to improve slightly after a quinsy 13 months after admission, showing more powers of control. However, two months later she was as bad as ever, developing much crying, frequently throwing her meals on the floor or fire, and even getting out of bed at night and pinching and biting another child who was asleep. Many uncontrollable actions then took place daily. She jumped suddenly out of bed and hurled a marble locker-top through a window (she said she felt dizzy and did not know what she had done until she saw the damage); she struck without warning or reason a nurse who was bathing her; picked up a chair and broke a large window; hurled a chamber through a ward window; hit a stooping person hard on the head, etc. She would implore not to be made to sit near windows, "in case I smash something." Eighteen months after admission she developed a strong desire to have her head covered day and night, as it "stopped her from spitting." She was very fond of school but attendance was often impossible, as suddenly she would bang on her desk for a few moments with her fists and then rapidly fly at and strike another girl or the teacher. It would be over in a few moments, and she would then cry: "It's no good. I can't stick it any longer."

During her stay in hospital (up to the present day) she has remained impulsive and uncontrollable. She is never normal in behaviour for a single day. Her intelligence is normal. A recent Binet-Simon test shows her at least up to her age-standard, and a similar test done at the age of nine years gave her mental age as 11+. Physical development is normal. There was at one time some mutual masturbation with another girl. There is no nocturnal restlessness. During the last four months she has shown the development of slowly advancing Parkinsonism. She has a slight Parkinsonian mask, slight cog-wheel rigidity in the legs, and a somewhat Parkinsonian attitude and gait.

Case 3. Female, age 13. Prior to the onset of encephalitis the child was described as "very bright," quite natural, well-behaved, with no bad temper, and with no "tricks or habits." She was in good physical health, slept well and was of normal intelligence, was not a nervous child, and displayed no neurotic signs in infancy. She had an acute attack of epidemic encephalitis in March, 1924, with fever, diplopia and inversion of sleep rhythm. She was in bed four days only and, immediately after she got up, a change of character was noticed which grew rapidly more marked and was fully developed within the first month. She became quarrelsome and disobedient, screaming with passion when reprimanded, and in addition became restless and developed tics. She suffered from dribbling (? over-salivation) and bouts of noisy breathing when excited. She became abnormally affectionate, her parents describing her displays of over-affection as "sickening." No diminution in intelligence was noticed by the parents, who said that she was "if anything a bit quicker."

The patient was admitted to hospital in October, 1926. There was a faint trace of Parkinsonism in her attitude and gait. She was quarrelsome and easily upset and was very restless, being unable to sit or stand in any one position for more than a few moments. She showed a violent temper when she could not get her own way, would fight readily, and displayed an uncontrollable impulse to pinch and slap anyone who came near, which she would obey constantly even if she were hit back in return. Bouts of noisy tachypnœa would come on at any time, particularly when she was excited, but were commonest at night, when she was very restless and slow in going to sleep. She had an uncontrollable tendency to behave in an over-affectionate way. When she was not pinching or slapping them, she would like to sit with her arm round one of the other patients and longed for caresses. She tried to behave in the same way to the nursing staff. They described her

as liking to be petted and "craving for sympathy." She was quite conscious of her abnormal behaviour, regretted it and said that she could not help it. She could only be kept out of mischief by being put to bed. She showed signs of trying hard to maintain self-control. When reproved for over-affection she said, "I can't help it," and when reproved for other actions said, "I can't help it. I try to be good but I feel I must do it. I will try to stop it." She became keen on school and did comparatively well, trying very hard. Sometimes she would say, "Can I stay upstairs by myself until schooltime? I feel I am going to fight." Four months after admission slight Parkinsonian bradyphrenia with easy fatigability began to develop. Seven months after admission her power of control was definitely greater and not so much discipline was required. She has been trying hard and steadily improving ever since, but her Parkinsonism has been slowly advancing.

At the present time physical development is normal, there is no premature puberty, and she is not yet menstruating. Apart from a definite degree of bradyphrenia her intelligence is normal. There is a slight Parkinsonian mask, excessive salivation, an attitude of flexion, and a typical, though not severely marked, gait with little arm-swinging. There is an occasional tremor of the right arm, but muscle tone is normal in the limbs, and there is no hypothalamic involvement.

THEORIES OF PATHOGENESIS.

The theories that have been put forward are very varied. Some of them may here be mentioned.

Marshall considers that the patients suffer from psychomotor excitement similar to that occurring during an attack of acute mania of the manic-depressive type; Mapother, that their restlessness does not resemble acute mania, but that the symptoms represent a definite regression primarily due to affection of some part of the brain underlying foresight; Shrubsall, that the condition is due to a persistence of the mental upset of the acute stage of the illness as a habit or a psychoneurosis. Leahy and Sands vaguely believe that the cause is a chronic irritation and stimulation of nerve-cells by the resolving infection; Potts, that it is a chronic toxæmia, and Cloake, a depression of the highest cortical functions in particular, by a general toxi-infective state partly resembling the acute effects of a neurotoxin like alcohol. The latter also suggests 'psychic trauma,' such as putting the patient in a bath against his will.

These theories either are based on an interpretation of the clinical features with which we do not agree, or are too vague, making no satisfactory attempt to explain the distinctive pathology that must produce such distinctive signs. Thus we can find no evidence for regarding the condition as one of acute mania, functional nervous disease, or general chronic toxæmia. Occurring in an encephalitis, running such an unfluctuating course so similar in every case, and uninfluenced by the environment, the condition would seem to be due to an organic lesion of the brain. But we think that there are grounds for being more definite than the above theories about the latter. We have suggested above that the key to the abnormal behaviour is an increase in the affective accompaniment of primitive and instinctive tendencies, and we think that it is possible to indicate the organic lesion responsible for this.

We suggest that the organic lesion is an encephalitic one producing a loss of cortical inhibition over, or chronic excitation of, the optic thalami. The thalamus is the seat of affective life and the effect of loss of cortical inhibition over it by a destructive lesion producing thalamic release is well known. A similar effect could be produced by a chronic encephalitic focus producing chronic irritation and excitation of it. By either of these means an exaltation of the affective function of the thalamus would be produced. The affective response of the organism to any stimuli, mental or physical, would be increased and the condition that we consider exists in the encephalitic children with behaviour disorders would be produced. Their instinctive tendencies would have their normal affective accompaniments exaggerated and their dominance as suggested above would be produced. Our suggestion is supported by the known predilection of the encephalitic virus for the thalamo-striatal level on the one hand, and by the fact that owing to the preservation of memory and intelligence there can be no serious involvement of the cortex on the other. If it is agreed that behaviour disorders represent a primary disorder of affect of organic origin the lesion must be thalamic, with one very hypothetical exception mentioned below, as the thalamus is the centre of affect.

Further support for the theory that the lesion exalts thalamic function is forthcoming in that probably the thalamus in man still has some responsibility for the origin in itself of crude, primitive, instinctive behaviour. As McDougall says : " It has been shown that instinctive actions of the mammalia are chiefly dependent on the basal ganglia of the brain (especially the optic thalamus)." McDougall's long list of instincts is not accepted by all psychologists, but he says : " . . . there is good evidence that each of the principal forms of instinctive activity is especially dependent upon a particular small mass of nervous tissue within the basal ganglia." Each patient with behaviour disorder certainly displays favourite forms of primitive conduct ; one is predominantly over-affectionate, another aggressive and self-assertive, and so on. Perhaps with the furtherance of knowledge the lesions may be localisable in a particular part of the thalamic region from the clinical signs.

There are, of course, no gross physical signs of thalamic release in the cases of behaviour disorder, though compared with normal controls they all show much greater emotional reaction to pin-prick, deep-pressure pain, etc. A comparison of Case 3 with a case of unilateral thalamic syndrome described by Head and Holmes is interesting. The latter patient confessed that he had become more amorous since the attack : " I crave to place my right hand on the soft skin of a woman. It's my right hand that wants the consolation. I seem to crave for sympathy on my right side." What would his behaviour have been like if his lesion had been bilateral ?

A possible but very hypothetical cause of a disorder of affect producing a disorder of conduct is involvement of the hypothalamus, which occurs quite often in encephalitis, i.e., a lesion affecting the hypothalamic visceral centres.

The James-Lange theory is discredited but the following sequence in the generation of emotion has been suggested by Herrick. An intelligent process, i.e., a process of cortical association, occurs and involves the thalamic neurones and so gains an affective colouring. The hypothalamic visceral centres are activated and the usual visceral accompaniments of emotion take place. Afferent impulses from the sites of visceral change then ascend to the thalamus and reinforce the original emotion. Herrick calls this a " back-stroke " action. An invoivement of the hypothalamus might disturb this " back-stroke " action. Might a disorder of affect and so of conduct thus result ? Worster-Drought has recorded a case of moral imbecility associated with dyspituitarism apart from epidemic encephalitis.

Finally, cases of behaviour disorder may be contrasted with cases showing the bradyphrenia of the Parkinsonian syndrome. The bradyphrenia, something apart from the mere appearance of mental inertia due to the physical state, is characterised by a good intelligence with a subjective state of mental inertia and loss of interest or 'feelings.' It is worth while suggesting that this condition is produced by a cutting out of the mental life of much of the normal susceptibility to affective states and tendency towards instinctive forms of response which together make up much of the 'animal spirits,' spontaneity, and *joie de vivre* of a normal person. That is, it is suggested that bradyphrenia is the exact opposite to the mental state of cases with behaviour disorder and is due to a depression of thalamic function. A chronic irritative lesion of the thalamus might produce behaviour disorder and, when eventually the lesion becomes paretic and the thalamus succumbs, the condition might pass into bradyphrenia. On the physical side the general hyperkinesia of the cases of behaviour disorder can be contrasted with the bradykinesia of Parkinsonism.

SUMMARY.

The clinical features of juvenile behaviour disorders in chronic epidemic encephalitis are described, with three illustrative cases. The children have a preserved intelligence and clear insight into their condition, realising the error of their conduct. The latter is compulsive and consists of actions of a crude, primitive and instinctive type. Its compulsiveness is regarded as being due to an overpowering increase in the pleasure that normally comes of obeying instinctive tendencies and in the displeasure that normally comes of obstructing them. The liability of a general increase in the affective response to all stimuli to produce this effect in the child, where instinctive tendencies are normally so prominent, is pointed out.

The pathology is organic and is an encephalitic process, causing exaltation of thalamic function either by thalamic release through loss of cortical inhibition or by chronic irritation and excitation. This is supported by (1) the known incidence of the encephalitic virus on the thalamo-striatal level, and by (2) the

preservation of memory and intelligence indicating that the cortex cannot seriously be involved. Also, the thalamus probably has some action in the origination, in itself, of primitive and instinctive behaviour.

There is a possibility that a hypothalamic lesion may produce affective and consequently conduct disorder.

The mental condition in cases of behaviour disorder is contrasted with bradyphrenia, which, it is suggested, is due to depression of thalamic function.

The observations in this paper were made in the Post-Encephalitis Lethargica Unit of the Metropolitan Asylums Board, and I have to acknowledge with thanks the permission of the Medical Superintendent of the Northern Hospital to publish details of the cases.

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A CRITICAL REVIEW OF THE CEREBROSPINAL FLUID SUGAR.

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TECHNIQUE.

In the series of 151 readings submitted we have estimated the spinal fluid sugar content by the method of Folin-Wu, as modified for the spinal fluid after deproteinisation with tungstic acid. In the majority of cases the estimation was performed within one hour of the collection of the fluid, but in some instances the examination was unavoidably delayed, even up to as much as 12 hours after collection. Glycolysis is usually stated to occur rapidly in spinal fluids kept at room temperature unless the fluid has been previously deproteinised, and in cases where specimens have to be conveyed to a laboratory from outlying districts erroneously low readings may result. It may therefore be advisable in some instances, where scientifically accurate readings are desired, to deproteinise such specimens before transit to the laboratory. In our opinion glycolysis only occurs with sufficient rapidity to negative the clinical value of the estimation in fluids with a high protein content, and in fluids from cases of meningitis in which pyogenic bacteria are present, providing that not more than 12 hours elapses between collection and examination. As the majority of specimens received in a general laboratory do not fall, under ordinary circumstances, within either of these categories, we see no reason for discarding specimens as valueless because of some delay in transit. In support of our contention we append the results of duplicate sugar estimations on 14 unselected samples of spinal fluid. The sugar content was determined on a part of the specimen immediately upon its arrival at the laboratory. An untouched fraction of the same fluid was then allowed to stand for about 12 hours on the laboratory bench at room temperature, after which time its sugar content was again estimated.

Diagnosis.	*Protein in mgm. %*	*Sugar (1) in mgm. %*	*Sugar (2), after 12 hours, in mgm. %*
Uræmia	200 ..	167 ..	154
G.P.I.	125 ..	60 ..	57
G.P.I.	100 ..	72 ..	64
Epilepsy	100 ..	75 ..	74
Spinal Syphilis ..	75 ..	80 ..	76
Encephalitis	75 ..	91 ..	87

* From the University Laboratories, and the Biochemical Department of the Queen's Hospital, Birmingham.

Diagnosis.	*Protein in mgm. %*	*Sugar (1) in mgm. %*	*Sugar (2), after 12 hours, in mgm. %*
Undiagnosed	75	69	64
Disseminated Sclerosis	50	64	64
? Spinal Tumour ..	50	67	67
Cerebral Tumour ..	50	69	63
Sinusitis	50	71	71
Sinusitis	30	91	91
? Diabetic Coma ..	30	83	80
Congenital Syphilis ..	25	57	54

The protein content, as total protein, was estimated by Young and Bennett's[18] method on each specimen immediately after its arrival in the laboratory.

The number of observations on which to base an opinion is small, but we believe that glycolysis is not, in general, as rapid as it is frequently stated to be.

PREVIOUS OBSERVATIONS.

Greenfield and Carmichael[10] place the normal variations in the spinal fluid sugar from 45 to 85 mgm. per cent. They state that an increase is usually the reflection of an increase in the blood sugar, and that a hyperglycorrhacia is therefore to be expected in :—

1. Diabetes mellitus with hyperglycæmia. In these cases the spinal fluid sugar is commonly from 100 to 250 mgm. per cent. In diabetic coma figures from 300 to 600 mgm. per cent. have been noted.

2. Nephritis. In uræmia values of 145 to 200 mgm. per cent. have been obtained.

Apart from these two diseases, the authors state that it is probable that the spinal fluid sugar never exceeds 100 mgm. per cent. A slight increase may be observed in states of raised intracranial pressure from any cause. They also state that a decrease, amounting even to actual disappearance of sugar from the fluid, is seen in cases of tuberculous and ' purulent ' meningitis. In tuberculous meningitis the sugar values may range from 11 to 50 mgm. per cent. In ' purulent ' meningitis any values may be obtained from low normals down to complete absence.

Goodwin and Shelley[9] conclude that :

1. The spinal fluid sugar content is not constant for the same person.

2. There is no level within reasonable limits that might be considered normal for different individuals.

3. There is a definite relationship between the sugar content of the spinal fluid and the blood. This may be expressed as a percentage relation of the spinal fluid sugar to the blood sugar. The figure obtained in normal cases is fairly constant within the limits 45 to 65 per cent.

4. Ingestion of carbohydrates in sufficient quantity causes a definite and constant rise in the spinal fluid sugar, and disturbs the usual relationship between the spinal fluid and the blood sugar.

5. Meningitis, either tuberculous or 'purulent,' frequently gives rise to a low spinal fluid sugar reading, both as regards the actual content in the spinal fluid and in relation to the blood sugar.

Crawford and Cantarow [5], as the result of the study of 210 cases, state among other conclusions that "great increase in globulin and high sugar content are characteristic of increased intracranial pressure in general, and particularly of brain tumour. High sugar values are also obtained in various mental disorders. The essential cause of hypoglycorrhacia is glycolysis, which occurs to a large extent in acute suppurative meningitis, and to a slight degree in tuberculous meningitis. Sugar determinations are of great value in the differential diagnosis of tuberculous meningitis, especially from epidemic encephalitis."

Spurling and Maddock [14], using the Folin-Wu method of sugar estimation, found no significant variation in the sugar content of the spinal fluid in cases of brain tumour. The sugar values varied in their series from 57 to 84 mgm. per cent., with an average of 74 mgm. per cent.

Halliday [11], estimating the spinal fluid and blood sugar by MacLean's method, concludes that :

1. In epidemic encephalitis the fasting level of the blood and the spinal fluid sugar is within normal limits, the fasting level of the spinal fluid sugar being lower than that of the blood sugar.

2. Associated with the physiological blood sugar curve is a corresponding spinal fluid sugar curve. The latter is a delayed curve, and the non-recognition of its existence may have been responsible for the high spinal sugar findings obtained by many workers in encephalitis and other allied conditions.

3. A specimen of spinal fluid to be estimated for its sugar content should be collected after a 12 hours' fast, coincidently with a sample of blood for a similar investigation. If the sugar content of the spinal fluid is normal its ratio to the blood sugar should lie between 50 and 70 per cent.

4. Specimens of spinal fluid at varying intervals after a meal give sugar readings which are valid only when these readings are correlated with the complete sugar curves, both of the blood and spinal fluid.

In a series of 25 cases, of which 20 were cases of encephalitis, Halliday obtained the following figures for the blood and spinal fluid sugar, estimated on specimens taken after a 12 hours' fast. The blood sugar varied from 68 to 128 mgm. per cent., the average figure being 95 mgm. per cent. These figures correspond with those given by MacLean, 80 to 128 mgm. per cent., as normal for the fasting level in a healthy person. The spinal fluid sugar ranged from 42 to 70 mgm. per cent., the mean being 56 mgm. per cent.

Cookson[5], in a small series of readings, found that " in almost every case diagnosed finally as encephalitis lethargica the sugar content was definitely increased above the normal, and that in no instance was there any diminution."

Bourges, Foerster and Marcandier[3], quoted by Halliday, in a series of 6 cases of epidemic encephalitis, found a hyperglycæmia associated with a hyperglycorrhacia. This increase they considered to be a valuable aid in diagnosis. The methods of estimation employed were not stated, and they accepted as the normal sugar content of the spinal fluid Mestrezat's figure of 53 mgm. per cent.

Foster[6], quoted by Halliday, using the Folin-Wu method, and accepting for the average normal spinal fluid sugar reading the figure of 53 mgm. per cent., obtained values ranging from 53 to 113 mgm. per cent., with an average of 76 mgm. per cent., in a number of fluids examined. He concluded that the spinal sugar was increased in encephalitis, but he could not find any corresponding increase in the blood sugar.

Thalheimer and Updegraff[16], quoted by Halliday, using Benedict's method of sugar estimation, found both a hyperglycæmia and a hyperglycorrhacia present in encephalitis. Their figures, even for the normal blood and spinal fluid, are extraordinarily high. Most of the specimens were collected coincidently after 12 hours' starvation.

Foster and Cockrell[7], quoted by Halliday, using the Folin-Wu method, and considering the normal spinal fluid sugar to lie between 40 and 60 mgm. per cent., found readings over 60 mgm. per cent. in 34 out of 35 cases of encephalitis. Similar high values were obtained in other pathological states such as brain tumour, gas poisoning, septicæmia and tabes. They considered that the increase in the spinal fluid sugar in encephalitis, though not confined to that condition, was useful in making a diagnosis.

Coope[4], using the Folin-Wu method, examined 11 cases of encephalitis and found 9 with a spinal fluid sugar over 60 mgm. per cent., but of 69 specimens from other diseased states, mainly insanities, 60 also gave values over that figure. He concluded that " the French tendency to regard a high sugar content of the spinal fluid as in favour of lethargic encephalitis does not appear to be justified, as figures quite as high occur in other nervous diseases."

Stowe[15], in a series of 122 cases, found that 15 cases of neurosyphilis gave values of 60 to 91 mgm. per cent., thus showing no departure from the normal, since he places the normal limits of variation of the spinal fluid sugar between 60 and 90 mgm. per cent. Of 20 cases of encephalitis lethargica only two showed any abnormality; in both cases the figure was high. Twenty-one cases of proved tuberculous meningitis had a sugar content varying from 10 to 50 mgm. per cent., and 14 cases of purulent meningitis yielded values ranging from 0 to 25 mgm. per cent.

Alpers, Campbell and Prentiss[1] examined 421 spinal fluids from normal cases, epidemic encephalitis, general paresis, dementia præcox, manic-depressive insanity and various miscellaneous conditions. They employed the Benedict-

Osterburg method for the estimation of the urinary sugar, modifying it to suit their own particular requirements. A control estimation was made in each case by the Folin-Wu method. They placed the normal range of the spinal fluid sugar between 50 and 65 mgm. per cent. In epidemic encephalitis the average figure obtained was 82 mgm. per cent., an increase which they consider to be of some diagnostic value. Twenty-one cases of dementia præcox yielded an average figure of 80 mgm. per cent., but high readings were less often obtained than in the cases of encephalitis. In general paresis and manic-depressive insanity the sugar readings fell within the normal limits of variation.

Purves-Stewart[13] states that an excess of sugar is often found in the spinal fluid in cases of epidemic encephalitis, in marked contrast with the two chief conditions with which the disease is likely to be confused, viz., tuberculous meningitis and early poliomyelitis.

Fremont-Smith and Dailey[8] conclude that the cerebrospinal fluid and the blood contain reducing bodies other than glucose. Sugar tolerance curves in the blood are followed after a latent period by a similar variation in the spinal fluid, and the spinal fluid sugar may be at one stage higher than the blood sugar. Spinal fluid sugar values above 80 mgm. per cent. occur in a great variety of conditions beside encephalitis lethargica. Such high values are usually associated with a hyperglycæmia, which is a not uncommon concomitant of cerebral lesions. In the absence of a hypoglycæmia, spinal fluid sugar readings below 50 mgm. per cent. nearly always signify acute infection of the meninges. Values between 50 and 80 mgm. per cent. occur in the normal, in many metabolic diseases, in acute meningeal infections, encephalitis lethargica, brain tumour and syphilis, etc. The authors consider that the spinal fluid sugar should be determined on fasting patients and compared with the coincident blood sugar.

Wilcox, Little and Hearn[17] conclude that the spinal fluid sugar should always be estimated in relation to the blood sugar. The normal relative value varies from 40 to 60 per cent. of the blood sugar. High relative values are found consistently in epidemic encephalitis, meningismus, convulsions, acidosis and nephritis, in poliomyelitis of the bulbar type, and in a certain number of so-called normal cases. From the variety of cases in which a high relative reading is obtained it is evident that this is not a reliable diagnostic test. Low relative values are encountered in acute purulent and tuberculous meningitis with great regularity, and this finding is of considerable diagnostic importance.

Levinson[12] concludes from the study of a small series of fluids that, for clinical purposes, there is no especial advantage to be derived from the determination of the sugar values on coincident samples of blood and spinal fluid. He found that the sugar content of various meningitic fluids examined ranged from 45 mgm. per cent. down to a mere trace, that the figures varied from day to day, and that the spinal fluid sugar bore no direct relation to the blood sugar, which, in the same series of cases, ranged from 84 to 176 mgm. per cent.

DISCUSSION.

A review of recent work on the spinal fluid sugar and its diagnostic value reveals, amid many contradictions, a general agreement to the effect that the findings are constantly lower than normal in tuberculous meningitis, and that there is a further constant reduction in all cases of purulent meningitis, irrespective of the particular micro-organism responsible for the lesion. With regard to the diagnostic value of increased sugar readings, it is generally accepted that any state of hyperglycæmia is followed after an interval by a rise in the spinal fluid sugar content, but, in the absence of a hyperglycæmia, it appears doubtful whether high spinal fluid sugar values ever occur with sufficient consistency to be of any diagnostic significance. A hyperglycorrhacia, without an accompanying hyperglycæmia, has been inconstantly observed in cases of brain tumour, epidemic encephalitis, and various other lesions of the central nervous system. We consider, from a brief review of the recent work on the subject, that there is no generally substantiated evidence that the spinal fluid readings, either alone, or in relation to coincident blood sugar readings, are of diagnostic value in epidemic encephalitis.

In our opinion, some of the contradictory observations upon the importance of the spinal fluid sugar figures in encephalitis may have arisen as the result of the differing values given by the various sugar-estimation methods in vogue. Estimations, by the same worker, of the sugar content of a single specimen of spinal fluid, performed in duplicate by the methods of MacLean and of Folin-Wu, yield almost invariably, in our experience, higher readings by the former method than by the latter. The same observation also holds true for the blood sugar. We are not prepared to discuss the relative accuracy of the various methods in use, but we do suggest that the confusion is liable to continue until such time as a single standard method of sugar estimation is in universal use. In addition there must also be a universally accepted normal range of variation of the spinal fluid sugar.

Recent workers, with the exception of Levinson, stress the importance of estimating the sugar content on coincident samples of blood and spinal fluid in order to obtain the spinal fluid—blood sugar ratio. While this ratio may be of scientific interest, it does not appear to us to confer upon the clinician any decided advantage over the actual spinal fluid sugar reading. The various investigators are not in complete accord as to the exact normal limits of variation of this ratio, and it has yet to be shown that the ratio presents a sufficiently constant figure for any one particular disease to warrant its routine application. There is ample confirmation that actual diminution in the spinal fluid sugar finding is significant of a meningitic lesion, of either tuberculous or pyogenic origin. In these diseases the blood sugar is usually not affected, and it exercises no influence upon the diagnostic significance of the spinal fluid figures per se.

It is, however, obviously of importance, in view of the demonstration of physiological sugar curves in the cerebrospinal fluid, to collect the specimen whenever possible after a period of fasting. In hospital practice this precaution can easily be ensured by collecting all the samples in the morning, after the night's fast, and before any further food has been taken. It is seldom that a case is so urgent that this procedure could not be adopted as a routine measure in hospitals and similar institutions. In private practice there are difficulties in the way of adopting this procedure, and it would therefore be of advantage in some private cases to take coincident samples of the blood and spinal fluid for sugar determinations.

Taking the figures 50 to 80 mgm. per cent. as the possible normal limits of variation in the spinal fluid sugar (figures arrived at after a study of the normality ranges advocated by the authorities previously quoted), we have encountered high sugar values in such diverse conditions as uræmia, cord tumour, congenital mental deficiency, hysteria, brain abscess, cerebral hæmorrhage, acute encephalitis, general paresis and disseminated sclerosis. In the accompanying Table we have plotted down the sugar values obtained in our series of estimations. A glance at the Table shows that the majority of the readings fall within the normal limits of variation, and that in only two diseases do the spinal fluid sugar values lie constantly and definitely outside the normality range. In both these conditions, tuberculous meningitis and acute purulent meningitis, there is a marked reduction in the sugar content of the spinal fluid. All the cases of tuberculous meningitis were proved either by the bacteriological finding of the tubercle bacillus in the cerebrospinal fluid, or by the pathological findings at the subsequent autopsy. In the majority of the cases both the bacteriological and the histological findings were available. Similarly, all the cases of acute purulent meningitis were proved by the isolation and cultivation of the infecting bacterium during life. In many of the cases the confirmative autopsy finding was also available.

In four of our cases of acute purulent meningitis and in two cases of tuberculous meningitis the spinal fluid sugar fell below 10 mgm. per cent., and in three of former cases it was probably entirely absent. There is a simple and satisfactory explanation for the reduction in the spinal fluid sugar in acute purulent meningitis, viz. that the bacteria present utilise the sugar in the course of their growth and multiplication, just as they readily utilise glucose in artificial culture media. It is doubtful whether this explanation is applicable in the case of tuberculous meningitis, since the tubercle bacillus does not evince any especial partiality for glucose in artificial culture. In connection with this fact it is significant that the spinal fluid sugar rarely drops below 10 mgm. per cent. in tuberculous meningitis.

All observers agree that a constant, definite hypoglycorrhacia occurs in association with a tuberculous or pyogenic infection of the meninges, and

that, taken in conjunction with positive clinical evidence, a definite reduction in the spinal fluid sugar content is diagnostic of the meningitis. Our findings add further confirmation to the generally accepted opinion.

It will be seen from the Table appended that we have not observed the high sugar values in encephalitis lethargica that some other workers claim to have obtained. In our experience a hyperglycorrhacia does not occur with any constancy in encephalitis lethargica, providing that the upper limit of the normal variation in the spinal fluid sugar is placed at 80 mgm. per cent. Had we accepted the figure of 53 mgm. per cent. as the normal, it is obvious that our findings would have led us, erroneously we believe, to assume that a hyperglycorrhacia was present in encephalitis, though its significance would have been negatived by a similar finding in other diseased conditions of the central nervous system. It has been suggested by certain observers that the finding of a hyperglycorrhacia is an important aid to the diagnosis of encephalitis lethargica. In our opinion the finding of a normal or high spinal fluid sugar is a valuable aid in the elimination of tuberculous meningitis, when a diagnosis of encephalitis lethargica is under discussion, but otherwise we do not believe that the estimation of the spinal fluid sugar offers a trustworthy short-cut to the diagnosis of the latter condition.

We regret that we have not had an opportunity of examining fluids from cases of syphilitic meningitis, or acute anterior poliomyelitis, since the readings, especially in the latter condition, would undoubtedly have been of interest.

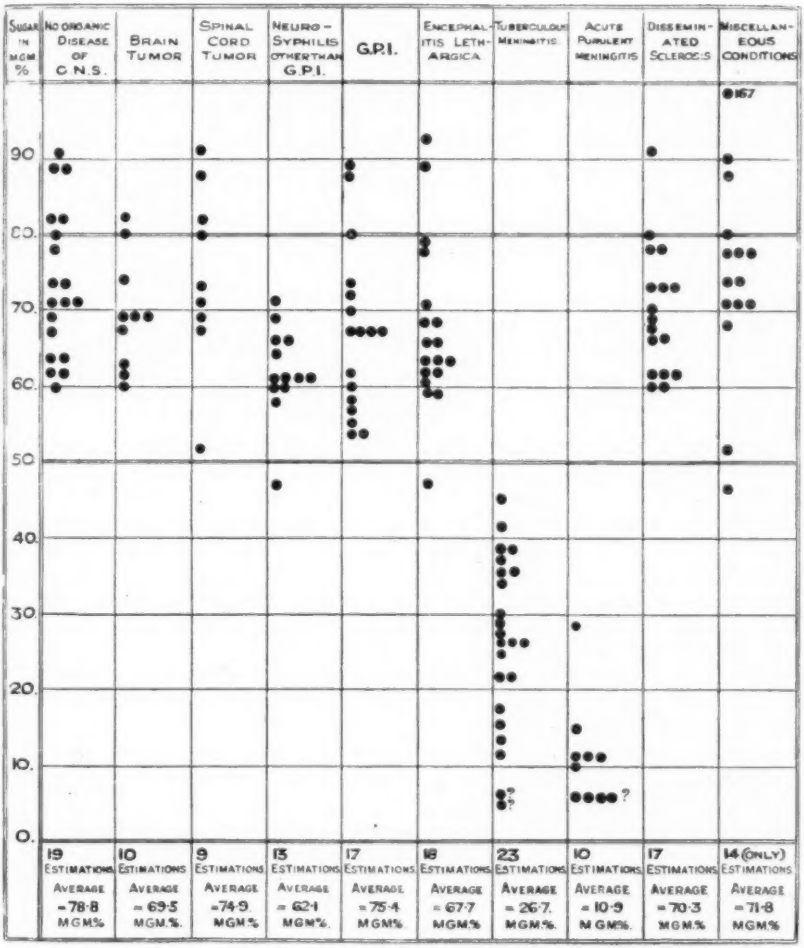
SUMMARY.

1. One hundred and fifty-one estimations of the cerebrospinal fluid sugar have been performed, by the Folin-Wu method, on fluids from various diseased states of the central nervous system, and on fluids from individuals showing no evidence, clinically, of any involvement of the central nervous system.

2. The results obtained confirm the opinions of previous observers that a constant reduction, which possesses diagnostic value, is encountered in tuberculous and acute purulent meningitis.

3. A constant hyperglycorrhacia has not been observed in encephalitis lethargica, and the finding of an increased spinal fluid sugar is not believed to be significant of that condition.

We are indebted to the courtesy of Dr. P. Hughes for the spinal fluids from known paretic inmates of the Barnsley Hall Asylum, Bromsgrove. We also desire to thank the Honorary Staffs of the Queen's Hospital, and the Birmingham and Midland Hospital for Nervous Diseases, for the material and facilities that they have willingly placed at our disposal.


Sugar in mgm %
No organic Disease of C.N.S.
Brain Tumor
Spinal Cord Tumor
Neuro-Syphilis other than G.P.I.
G.P.I.
Encephalitis Lethargica
Tuberculous Meningitis
Acute Purulent Meningitis
Disseminated Sclerosis
Miscellaneous Conditions
167
90
80
70
60
50
40
30
20
10
0
19 Estimations Average = 78·8 mgm%
10 Estimations Average = 69·5 mgm%
9 Estimations Average = 74·9 mgm%
13 Estimations Average = 62·1 mgm%
17 Estimations Average = 75·4 mgm%
18 Estimations Average = 67·7 mgm%
23 Estimations Average = 26·7 mgm%
10 Estimations Average = 10·9 mgm%
17 Estimations Average = 70·3 mgm%
14 (only) Estimations Average = 71·8 mgm%

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THE QUANTITATIVE ESTIMATION OF SUDORIFEROUS ACTIVITY AND ITS RELATION TO TEMPERAMENT.

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INTRODUCTION.

TEMPERAMENT, as defined by McDougall[1], is the sum of the effects on the mental life of the metabolic or chemical changes that are constantly going on in all the tissues of the body.

Temperaments have been roughly divided[2] into four large groups: Sanguine, Choleric, Phlegmatic and Melancholic. In view of more recent work[3] on the mutual balance of endocrine activity, it has been thought possible to correlate physical activities and characteristics with temperament. Clouston[4] in 1904 remarked the doctrine of temperaments and diatheses. Kretschmer[5] has attempted a selection of certain physical types as found amongst the mentally abnormal. The authors hereunder endeavour to adopt the above classification of temperament and, applying it to the 'sane' and 'insane,' seek for its correlation with one particular physical characteristic. The two former of these groups are stated by Stoddart[6] to possess rapidity of thought and ease of receptivity, the two latter to possess a slower degree of thought and receptivity.

The observation of physical characteristics must to some extent at least be influenced by subjective criticism, and it is thought that observations made on a quantitative rather than a qualitative basis are free from this possible error. The correlation between mental type and physical states and reactions has been the subject of investigation at Parkside Mental Hospital for some time past. Some tentative conclusions have already been arrived at concerning a correlation between mental type on the one hand, and on the other (*a*) blood sugar at rest[7]; (*b*) superficial vascular response[8]; (*c*) peripheral circulatory activity[9]; and (*d*) the response to thermal stimulation[10]. Following on these, and particularly in view of the conclusions reached by the last-mentioned investigations, which showed a widely divergent sudoriferous response after external heat changes, an attempt is herewith made to place on an objective basis the correlation of mental type and temperament with one particular evidence of metabolic and chemical change, viz. loss of moisture from the skin.

Popular belief has it that the possessor of a firm and warm handshake is a man of buoyant and energetic type, whereas the owner of a cold and clammy palm is of underactive and introspective tendencies. It is recognised clinically by writers on psychiatry that the primary dement has a moist and cyanosed hand, whereas the maniac has a hand which, though also moist, is of a raised temperature.

TECHNIQUE.

In small part for these reasons, and in large part for more practical reasons, the hand up to the level of the wrist has been chosen for particular investigation regarding moisture-loss at rest. The apparatus employed and certain references to the quantity and activity of the sweat glands of the hand have already been described elsewhere[11]. In brief, a current of thoroughly dried air is drawn over the hand of an individual who has been kept at physical and mental rest for some period previous to the experiment, constant experimental conditions obtaining as far as possible. The air after passing over the skin of the hand is drawn through previously weighed tubes of sulphuric acid and the increase in weight is taken as indicating, at all events for comparative purposes, the sudoriferous loss from the hand examined, increases being measured in grammes per period of ten minutes.

PERSONAL OBSERVATIONS.

In order to obtain some estimate as to average moisture-loss amongst the mentally healthy a series of seven males and twenty-five females (nurses) was examined (Section A). Following on this (Section B) thirty cases of primary dementia were examined. An attempt was made (Section C) to find an equal number of cases of pure mania, but unfortunately only five have been found to date which could be controlled sufficiently to allow of the performance of the experiment with accuracy and with safety to patient and apparatus alike. The hand of the imbecile has been found to resemble the hand of the primary dement in respect of its clamminess, and for this reason (Section D) a series of twenty-three cases was examined. Finally, in view of the stated diminution of sweat secretion in melancholia, fifteen cases of this were also subjected to examination (Section E).

The exact figures obtained (grammes in 10 minutes) were as follows—an average being taken in cases where two or more estimations were made :—

Section A. Normal healthy male adults. Age 20—40. ·60, ·35, ·34, ·28, ·25, ·27, ·36. Average ·325.

Normal healthy females of similar age. ·315, ·180, ·330, ·400, ·510, ·425, ·390, ·495, ·355, ·160, ·220, ·150, ·190, ·265, ·240, ·280, ·500, ·280, ·310, ·265, ·180, ·145, ·215, ·130, ·175. Average ·29.

Section B. Primary dements. Age 20—40. ·15, ·13, ·12, ·15, ·07, ·14, ·13, ·10, ·08, ·14, ·11, ·07, ·11, ·26, ·10, ·22, ·18, ·17, ·08, ·10, ·16, ·09, ·14, ·11, ·15, ·10, ·14, ·18, ·22, ·18. Average ·103.

Section C. Maniacs. Age 20—40. ·24, ·26, ·87, ·46, ·19. Average ·40.

Section D. Imbeciles. Age 20—40. ·35, ·35, ·15, ·47, ·18, ·10, ·23, ·18, ·18, ·17, ·27, ·17, ·17, ·46, ·19, ·30, ·61, ·17, ·40, ·27, ·17, ·12, ·18. Average ·25.

Section E. Melancholics. Age 20—40. ·19, ·135, ·33, ·112, ·24, ·18, ·325, ·420, ·176, ·23, ·24, ·10, ·27, ·20, ·36. Average ·23.

It is seen from the above figures that whereas in the dementia præcox group there is a consistently low figure in all cases, the other groups show a very wide variation indeed, and a search must be made for some factor other than one of gross mental disease before discussion as to the etiology of variations in sudoriferous activity is entered into.

In a single healthy adult many factors influence the amount of perspiration lost by the hand in a given period. The greater number of these are obvious and are well recognised: external temperature, relative atmospheric humidity, amount of clothing worn, and recent physical effort. These conditions were, however, kept nearly constant in the above examinations and exerted but little influence on the findings.

It was, however, early realised in the experiments that individuals of different temperament may be found included within the same mental type group. It was, therefore, decided to ask the Assistant Matron, from her personal and intimate knowledge, to classify the twenty-five nurses into those who were of quick mentality, emotional and restless, and those who were, by contrast, phlegmatic, steady and even dull; also to make a group of those who were intermediate in type. A similar but independent classification judged from capacity and quickness in duty was made by the A.M.O. in charge of the wards where these nurses were employed. A classification was made as follows:

Group A, where both judges placed the individuals amongst the emotional, restless or mentally quick type.

Group B, where both agreed as to 'average' type or where judges disagreed.

Group C, where both judges were of the opinion that individuals were phlegmatic and steady.

The results were as follows, taken from the readings of the females of Section A above:

Group A. ·315, ·180, ·330, ·400, ·510, ·425, ·500, ·390, 4·95, ·355, ·160. Average ·370.

Group B. ·220, ·250, ·10, ·265, ·240, ·280, ·310, ·280. Average ·257.

Group C. ·265, ·180, .145, ·215, ·130, ·175. Average ·185.

This shows a very close correlation of temperament and sudoriferous activity, the mentally quick, emotional and restless group giving a figure exactly twice that of the steady and phlegmatic group. An intermediate figure is given by the average group. It is to be noted that only two out of the

eleven in Group A have a figure near that of the average of Group C, and that the latter has only one individual with a figure approaching even the average of Group B.

Encouraged by the above, we conducted an investigation on similar lines amongst the imbeciles. The A.M.O. in charge of the wards and the head attendant jointly classified the imbeciles into a loose grouping of 'restless' and 'apathetic.' Here again a close correspondence was noted: the former group gave figures of ·35, ·35, ·47, ·18, ·23, ·18, ·18, ·27, ·19, ·30, ·61, ·17, ·40, ·27, ·18, ·46—average ·30; and the apathetic group, ·15, ·20, ·10, ·17, ·17, ·17, ·12, ·17—average ·15. In this series also the marked difference in reaction between the two temperamental types was in evidence.

When a subdivision of dementia præcox cases, in which the figure appears to be almost invariably low, was made the following results were obtained: Katatonics (15), average ·131; hebephrenics (7), average ·136; dementia paranoides (5), average ·14; simple (3), average ·120.

It was not found possible to apply the above-mentioned classification of temperament to the melancholic group, though it was found that the cases which were noted at the time of examination to be markedly agitated gave figures of ·135, ·33, ·29, ·420—average ·28, which is slightly higher than the total average.

DISCUSSION.

Even from a casual examination of the above figures it is clear that there is a close correlation between temperament and sudoriferous activity. Assuming that temperament is the result of metabolic changes constantly occurring in the body, then the specious explanation of the findings is that an active physical and mental life, even though running in pathological channels, results in greater heat production. This necessitates a larger sudorific activity to retain the same temperature of the organism by evaporation. On the other hand, the secretion of sweat under constant experimental conditions may be looked on as an index of sympathetic activity. Developmentally the sympathetic is intimately related with the adrenals and thus with the endocrine system. Therefore, instead of regarding the increased secretion of moisture as a matter of cause and effect, one can think of its causation as correlated with an active endocrino-sympathetic system—the increased moisture-secretion and the active mental and physical life *both* being the results of a particular type of endocrine balance.

The imbecile group and the normal group are thus brought into line, while the findings in the melancholic group are by no means contradictory. In the primary dement we have almost invariably a reduced peripheral temperature with capillary and venous stasis. According to McWilliam[12] such cases have an under-excitability of the sympathetic nervous system. Thus there is less evaporation of moisture from the hands and therefore a paradoxical increase of sweating.

SUMMARY.

1. An attempt is made to correlate sweat loss with mental disease.

2. With the exception of primary dementia, and possibly mania, no such correlation is apparent.

3. A close correlation exists between temperament and sweat loss in the mentally healthy.

4. A close correlation exists between temperament and sweat loss in cases of imbeciles who have been roughly classified as 'restless' and 'apathetic.'

5. Primary dements show generally a decreased peripheral moisture loss.

6. The secretory activity of the sudoriferous glands of the hand is not decreased in melancholia.

7. The sudoriferous activity of the hands in mania appears to be increased.

8. The above changes may be explained on the basis of a need for heat dispersal as a direct consequence of increased heat production, or they may be regarded as changes coexistent with temperamental variations in response to a primary alteration of metabolic activity.

Our best thanks are due to Dr. H. Dove Cormac, Medical Superintendent of Parkside Mental Hospital, for permission to investigate and report on the above cases.

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SOME TENTATIVE CONCLUSIONS ON THE BOLTZ TEST IN GENERAL PARALYSIS OF THE INSANE.

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INTRODUCTION.

Results from the Boltz test have been published from time to time during the past three years, but so far the different investigators have seemed singularly at variance concerning their conclusions, although between them the total number of cases of general paralysis reported upon amounts to some 220.

Earlier observations from this hospital were published by Dr. J. P. Steel and myself [5] in 1926, but now that the test has been used here for just over two years the time seems to have come when something a little more definite might be said about it, especially as regards some points raised by other authors.

Most observers agree that the test is generally positive in general paralysis, some would even say invariably [1, 2, 3, 6, 7, 10]. At the other extreme are some who think it is positive in most fluids even apart from cerebrospinal syphilis [8, 11]. It has been suggested [2] that the test follows the globulin reaction in its results, and that it alters in company with the others after malarial therapy [3]. On the other hand one observer [4] says it remains positive after malaria.

NATURE OF CASES.

The observations that follow are based on a series of 346 Boltz tests performed on 267 patients, of whom 108 were cases of general paralysis. Of these 108, 56 were investigated by the Boltz test before malaria only, 21 before and after malaria, and another 31 after malaria only. The technique of he test is simple and has already been often described by others.

For comparison the general results of the globulin test were obtained from a rather larger series, viz., 672 tests on 496 cases, of which 180 were general paralytics, 126 tests being performed after therapeutic malaria. (See Table I.)

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TABLE I.

	Nature of Cases.		Lange and Globulin.				Boltz's Acetic Anhydride.			
			Cases	Tests	Glob. +	Glob. —	Cases	Tests	Boltz +	Boltz —
	Before Malaria.*		179	187	182	5	77	77	72	5
G.P.I.	After Malaria	Less than one year. of above*	44	57	38	20	21	23	29	6
		Less than one year. others	1	1			9	12		
		More than one year. of above*	38	68	29	39	5	6	21	23
		More than one year. others	—	—			25	38		
		Total after Malaria. of above*	68	125	67	59	21	28	50	29
		Total after Malaria. others	1	1			31	51		
	Total for G.P.I.		180	313	249	64	108	156	122	34
	Non-G.P.I.		316	359	32	327	159	190	1	189
	Grand Total		496	672	281	391	267	346	123	223

RESULTS.

On examining the tabulated results, two facts at once become obvious. Firstly, the Boltz test does not always agree with the globulin test; this is evident from the illustrative cases that have been chosen as fairly typical of the series (Table II). Secondly, it is not positive in the majority of fluids. In only 0·5 per cent. of non-G.P.I. fluids was it positive (one case), whereas the corresponding figure for the globulin series was 9 per cent.

In untreated paralytics the test was found negative in 6·5 per cent. of cases, but the globulin was so in only 2·75 per cent. This might perhaps be due to the Boltz test's only appearing positive later in the development of the disease than the globulin, especially since in several of these cases it was proved that the test became positive later even though malaria had been induced in the meantime. (This might be correlated with the behaviour of the test in fluids that first improved and then relapsed, as described below.) See Cases 133, 165.

Reference to Table I will show that the test certainly does become negative after malaria (See Cases 20, 53), but sometimes remains positive throughout several examinations (Case 100). But whereas the number of globulin tests thus becoming negative is 47·8 per cent., only 36·7 per cent. of the Boltz follow suit.

It is interesting to compare the relative rate of change of these two tests, by contrasting the results obtained within one year of malaria with those to be found more than a year after. Within one year, 34·5 per cent. of the globulins are negative as against 14·7 per cent. negative Boltz; after one year, however, the respective percentages are 57·4 and 52·3, showing that the Boltz test changes more slowly at first but tends to catch up the globulin results later. (See Cases 17, 57.)

TABLE II.

Illustrative Cases.

	Before Malaria.			After Malaria.			
Case Number	Lange.	Globulin.	Boltz.	Months since Malaria.	Lange.	Globulin.	Boltz.
133	34555,43300	+	—	2	23455,33200	+	+
165	55555,43310	+	—	2	14555,43000	+	+
				8	24544,31000	—	+
20	55555,42222	+		19	11555,43100	+	
				25	00321,10000	—	—
				42	02344,43210	—	—
53	55554,31000	+		3	00114,55400	+	
				11	01123,32100	—	
				17	01122,21000	—	—
				33	00011,10000	—	—
100	55555,54310	+	+	1	55555,55543	+	+
				6	55555,55443	—	+
				16	55555,55432	+	+
17	55555,54322	+		20	34555,43100	—	
				26	13345,52100	—	+
				40	12332,11000	—	—
57	55555,51100	+		14	00233,21100	—	
				20	00033,20000	—	+
				30	00011,10000	—	—
132	55555,55431	+	+	10	34555,44321	—	—
				21	03555,54320	+	—
108	55555,54330	+		2	35555,42200	+	+
				6	34455,43100	—	—
				20	55555,54321	+	—

It is, moreover, of interest to see how after both tests have been negative for some time the Boltz may remain negative even though the globulin again becomes positive. (Cases 132, 108.)

CONCLUSIONS.

Although nothing categorical can as yet be said about this test, the following tentative conclusions may be advanced.

1. The Boltz test is not usually positive in the cerebrospinal fluid except in presence—and almost exclusively in presence—of general paralysis.

2. The test does not always agree with the globulin test and may be positive when the latter is negative, and vice versa.

3. A positive Boltz test is almost invariably an indicator of general paralysis.

4. A negative Boltz test is not a reliable indicator of the absence of general paralysis, at least in the earlier stages.

5. The test tends to change after malarial treatment but not as frequently as the globulin one.

6. When the test does alter, it does so less rapidly than the globulin test.

7. When a recession of an improved fluid occurs the Boltz test tends to remain negative for a longer period than the globulin reaction.

I am indebted to the Medical Superintendant, Dr. F. M. Rodgers, for permission to quote hospital material.

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THE OCCURRENCE OF AMYOTROPHIC LATERAL SCLEROSIS IN CHILDREN.

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The following case came under our observation at the Mineral Water Hospital, the patient having been admitted for stiffness and aching in the lower limbs.

A.L., a Jewess, age 28. Occupation, dress finisher. There was no history of nervous or mental disease in her family. She was the third child of a family of six, all the rest being healthy.

She had had no illnesses to note until the age of ten, when she suddenly developed difficulty in walking. The trouble seemed to be chiefly in the right hip. She stayed in bed for four weeks, after which she returned to school but was eventually transferred to a school for cripples, where she remained for four years. She was able to walk considerably further than she can now, but frequently fell down. Since then the difficulty in walking has slowly and steadily increased. At the age of 23 she noticed that her hands were beginning to become weak and the muscles to waste. This condition has since progressed.

On examination she showed evidence of lower motor neurone disease of the upper extremities. Both thenar eminences, both deltoids and the muscles of the back were weak and wasted, but no fibrillary tremors were observed. There were no objective sensory changes, although the patient complained of some 'pins and needles' in the hands. In the lower extremities the symptoms and signs were those of upper motor neurone disease: markedly spastic gait with decided ataxia, increased tendon jerks, extensor plantar responses on both sides, patellar clonus on both sides. A contralateral response of the quadriceps was obtained from both patellar tendons. The abdominal reflexes were absent. She had slight precipitancy of micturition when she had a cold, but not otherwise, and the organic reflexes were not really interfered with. Objective sensory changes in the legs were absent, and posture sense was accurate, but she complained of some subjective paræsthesiæ.

She presented no signs or symptoms of bulbar paralysis or of any affection of the other cranial nerves, unless an unsustained indefinite nystagmus to the left and a slight tremor of the tongue unaccompanied by wasting or difficulty in speech or swallowing could be taken as such.

The Wassermann reaction was negative in both blood and cerebrospinal fluid and the latter presented no pathological features.

The blood count was as follows:—

R.B.C. 4,784,000. Hb. 90 per cent. C.I. ·95. Leucocytes 14,080. Polymorphs 50 per cent. Lymphocytes 34 per cent. Large mononuclears 11 per cent. Eosinophils 1 per cent. Basophils 4 per cent.

DISCUSSION.

The sudden onset of a spastic condition of the legs in a child of ten might suggest a condition such as myelitis, but the slow steady progression culminating in the progressive wasting of hand and shoulder muscles is against this, while on examination she presented the typical signs and symptoms of

amyotrophic lateral sclerosis and her case can be fairly diagnosed as such. The clear history from the age of ten, however, certainly constitutes a clinical rarity which merits consideration.

An examination of the literature of amyotrophic lateral sclerosis discloses a remarkably clear-cut picture of the clinical manifestations first described by Charcot[1] with his usual lucidity so long ago as 1865, but directly we pass from the clinical picture to the study of etiology and pathology we find an equally remarkable lack of uniformity of opinion. It is generally agreed that the three conditions which at one time were described separately, viz., progressive bulbar paralysis, progressive muscular atrophy and amyotrophic lateral sclerosis are all part of the same morbid process, since one may be added to the other in the history of the same patient and no definite sequence in this change can be demonstrated. Some observers have therefore described the condition as motor system disease, since motor cells and fibres whether belonging to the upper or lower neurones are involved, while other neurones of the central nervous system are either completely normal or only slightly affected by the morbid process. Most observers consider that this morbid process is a sclerosis; Naito[2], however, being convinced that the sclerosis was a definite sequel to inflammation, searched through the motor tract and found a patch of definite inflammation in the motor cortex, the rest of the cortex being little affected. He found that the pyramidal cells were considerably damaged and the cells of the lower layers also suffered; there was a glia reaction which was of an inflammatory character. Some vessels in the brain substance showed great perivascular infiltration and the infiltrating cells were nearly all lymphocytes. He was unable to find any similar patches in other parts of the brain or spinal cord, and concluded that an inflammatory process is responsible for amyotrophic lateral sclerosis and that the inflammation is due not to a toxin, but to an infection. In contrast to this Hassin[3] showed that the entire motor apparatus of the central nervous system excepting the nuclei of the ocular nerves is involved in a degenerative process. Warner[4] in a careful microscopical study of the condition concluded that the following changes could be found:

(1) Reactive pia-arachnoid changes.

(2) Marked degenerative changes in the cells of the anterior horns of the spinal cord, motor nuclei of the bulb and to a lesser extent in the motor area of the cortex; practically none in the occipital, temporal and frontal lobes or in the posterior horns of the spinal cord.

(3) Marked degenerative phenomena of some pyramidal tract fibres.

(4) Accumulation of lipoids in the adventitial spaces of the blood vessels of the entire central nervous system and especially in the areas of the pyramidal tract fibres.

(5) Absence of inflammatory appearances.

Thomson and Riddoch[5] state that changes similar to but less severe than those in the motor tract may be found in the spinocerebellar tracts; this was probably the case in our patient, since ataxia was a definite feature of her condition.

Grasset and Rimbaud[6] have described changes in the short association fibres of the cord, while Jelliffe and White[7] also mention degenerations in the lateral columns and Clarke's column. The frontal cortex may also be involved, as may be the basal ganglia.

From these considerations it is obvious that it is very difficult to define the exact pathological limitations of amyotrophic lateral sclerosis and that many cases described may differ considerably in their pathogenesis. Matzdorff[8] has sought to get over this difficulty by enlarging the conception of the syndrome and considers that the following may all be grouped together :—

A. Cases of exogenous origin due to toxæmia or infection. (1) The principally nuclear type : (*a*) chronic progressive bulbar paralysis ; (*b*) spinal muscular atrophy of the Duchenne-Aran type. (2) Mixed nuclear and tract type : (*a*) amyotrophic lateral sclerosis ; (*b*) chronic anterior poliomyelitis. (3) The principally tract type : spastic spinal paralysis.

B. A familial endogenous class of case : (1) of the nuclear type ; spinal muscular atrophy of the Werdnig-Hoffman variety ; (2) of the tract type : hereditary spastic paralysis.

When we come to the question of age incidence we find that all authorities are agreed that it is typically a disease of middle age, but there is no unanimity as to whether it can occur in children or not ; Oppenheim[9] describes it as a disease of middle life, observed only in isolated cases in childhood and then in brothers and sisters. He considers the average duration of the disease to be from two to four years, but admits that it may last longer. Similarly Jelliffe and White[7] state that it is known to occur in children but that it is most prevalent from the age of 30 to 40. They found that the average duration of over 100 cases was two years, but some have persisted for ten years and a few whose diagnosis was questionable for much longer.

Léri[10] thinks that any cases which have been reported as lasting for 10, 15 or 20 years must be cases of myelitis.

In 1905 Gordon Holmes[11] described two cases of family spastic paralysis in two sisters, ages 15 and 13 respectively, when they came under his notice. The lower limbs had been the seat of spastic paralysis from birth, the condition first being noticed when they tried to walk. Wasting of the small muscles of the hand had been noticed at the age of five in the elder sister and in the younger sister when she began to write. The picture on examination was similar in every respect to amyotrophic lateral sclerosis as met with in later life.

Analogous family cases have been described, for example by Gee.[12] A father of 37 and a daughter of 12 showed a condition simulating amyotrophic lateral sclerosis, while a son, age 11, had a spastic condition of both limbs, but no atrophy of muscles.

Ormerod[13] described a case of three brothers out of a Jewish family of 10 children, the eldest of whom, age 23, had spasticity of his lower limbs since the age of six, and the hands had been weak for three years. The small muscles of the hand and those of the legs were uniformally wasted. He also had nystagmus.

The next patient, age seven, had spasticity of the lower limbs but no affection of his upper limbs. A third, age four, had spasticity of the lower limbs and weakness of the extensors of the wrists and fingers.

Maas[14] described the cases of a brother and a sister. Both began to suffer from weakness of the legs about the age of 12. The arms became weak and speech was affected during the next few years. On examination at the age of 29 and 26 they showed characteristics identical with those of amyotrophic lateral sclerosis.

Hoffman[15] described four cases in one family in whom the affliction had first been noticed in childhood and who at the time of examination presented the symptoms of amyotrophic lateral sclerosis with some bulbar paralysis.

The cases of Higier[16], four sisters with amyotrophic and spastic conditions of the limbs, seemed different because they presented marked mental deficiency, while Seeligmüller's[17] group of four children in one family with some spasticity but universal muscular weakness hardly comes under the same class.

Other cases of a similar nature mentioned in the literature are as follows: Bogaert[18] described a case with sudden onset at age of 18 in the right lower limb. Within eight months all four limbs were affected. The first bulbar symptoms appeared less than a year after the origin. In 23 months the disease had advanced to its complete clinical picture and the patient died. The illness advanced by exacerbations with rises of temperature; ophthalmoplegia occurred in the last stages. This subacute course is unusual and resembles that of a progressive subacute encephalitis, but the pathological findings and spinal symptoms seem to point to amyotrophic lateral sclerosis.

Bouchaud's[19] case was that of a boy of 16 who presented a lateral sclerosis, of a hemiplegic type. The patient died at the beginning of the bulbar phase. The evolution was exactly the same as the sclerosis of adults.

Berger's[20] case was that of a child of 12 years, which began with paralysis of the motor branch of the fifth nerve; salivation then developed, with difficulty in swallowing and articulation. Facial paralysis especially on the right supervened and then affection of the pyramidal tracts of both sides.

Brown's[21] case was that of a boy of 15; the onset at the age of 12 was one of typical bulbar paralysis, with progressive bilateral affection of the upper face and dyspnœa. In some weeks atrophy of the arms of the face and of the neck set in, with fibrillary contractions of the whole musculature, exaggeration of tendon reflexes, contractures in the lower limbs, and with ankle clonus constant on the right and intermittent on the left. In addition the following authors have described cases of bulbar paralysis without pyramidal

involvement ; Hoffman[22], Thomson[23], Fazio[24], Remak[25], Trömner,[26] Marinesco[27]. Souques[28] describes a case in a girl of 19 with a two to three years' history. Bogaert[18] also describes a bulbar case in a boy of 10.

Certain familial cases of bulbar paralysis in the literature are not typical and show certain characteristics of pseudobulbar paralysis. In Souques' opinion the evolution of bulbar paralysis is much more rapid in the child than in the adult, although he admits there may be exceptions.

To sum up, cases occurring in children are referred to by several authors but in view of the uncertainty of the pathological limits of true amyotrophic lateral sclerosis it is obvious that caution must be exercised in accepting all such cases. Firstly, it may be recalled that two definitely established conditions are described in children which bear some clinical resemblance to the motor system disease in adults, viz. (1) the progressive muscular atrophy of infants, generally referred to as Hoffman and Werdnig's disease, and (2) family spastic paralysis ; neither of these probably has any pathological relationship to amyotrophic lateral sclerosis. Secondly, the late results of such definitely inflammatory conditions as polioencephalitis and myelitis may show a resemblance to the true motor system disease. As an example the following case which has recently come under the observation of one of us may be quoted.

> W.L., age 13, male. He was a healthy baby and there was no history of difficult labour or any paralysis at birth.
>
> He was vaccinated at three months. On the third day thereafter he was feverish, vomited, and suffered from a cough and a rash. During the next few days he had several fits.
>
> For three months after the onset of this illness he did not move his left arm at all, and when he began to walk, at two years, it was noticed that the left leg was abnormal and that he did not get his heel to the ground.
>
> On examination at the age of 13 he was found to be suffering from a mild degree of left hemiplegia ; the tendon reflexes were slightly increased on that side, especially the knee jerk ; ankle clonus and sometimes patellar clonus were elicited ; the abdominal reflexes were slightly weaker on the left side and the plantar reflex was doubtful. The muscles were in a state of mild hypertonicity with a certain degree of equinus which yielded to treatment.
>
> In addition, the left deltoid and scapular muscles showed marked wasting, lack of tone and fibrillary tremors, apparently being affected by a lower motor neurone lesion. These also improved markedly with treatment.

In this case of vaccination encephalitis, resembling one recently shown by Dr. J. P. Martin[29], both upper and lower motor neurones have been involved. These considerations make us view with considerable suspicion many cases described in the literature as adolescent amyotrophic lateral sclerosis and seem to indicate that true examples of this are of the greatest rarity.

So far as our own case is concerned there is no doubt that when the patient was admitted to hospital and came under observation she presented a reasonably typical clinical picture of amyotrophic lateral sclerosis, slowly progressing, with symptoms of involvement of the whole motor tract, while sensory manifestations were conspicuous by their absence. Purists would probably consider

that the sudden onset of the spasticity at the early age of 10 and the long duration of the condition—which has now lasted for 18 years without doing more than cripple the patient—debar us from describing it as a true case of Charcot's diasease. Yet, as has been shown in this study, the clinical syndrome is not so clearly defined as might be supposed and therefore the propriety of including some of these rarer examples under this nomenclature must be left sub judice.

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Short Notes and Clinical Cases.

NEUROSES IN THE TROPICS.

By H. LAING GORDON, Kenya Colony.

These notes are based upon observation in Kenya within one degree N. and S. of the equator, at an elevation of between 4,000 and 7,000 feet. The colony is little more than twenty years old. The total white population is 12,500, of whom about 5,000 are in the two townships, Nairobi and Mombasa; the remainder is scattered widely. The rate of increase is about 500 a year. The crude birth-rate, so far as can be ascertained, is about 18, the death-rate not more than 7 to 8 per thousand.

The neuroses of the whites in Kenya present no peculiarity but their prevalence is high and is attributed locally to tropical heat and light, to altitude, erroneous diet, isolated life, and to irritation from incompetent native labour. If these are genuine factors, too much importance may be given to them without a preliminary estimation of the psychic quality of the population. This estimate I have attempted from a limited standpoint in the *Kenya Medical Journal* for January, 1928. Briefly, experience has led to the conclusion that although the colony contains much of the very best mental and physical material for colonisation, it is receiving unchecked a high proportion (1) of immigrants in whom neuroses, psychoneuroses and psychoses already exist, either alone or along with nervous or other organic disease; (2) of others seeking the climate and open-air life to benefit their so-called neurotic constitutions or mental instabilities, as well as the usual flow to a young colony of chronic alcoholics; (3) of those termed 'public school failures,' and of unstable males and females seeking fashionable 'self-expression' in sport and pleasure. I believe these facts indicate the main source of supply of the neuroses in Kenya and account for the suspicion thrown upon the environment. From the point of view of the social psychologist they present a problem affecting colonial development and progress—one likely with the advance of psychological knowledge to involve the tropical specialist in new and important responsibilities for which preparation seems to be necessary.

This view of the main source of neuroses in colonies such as Kenya may receive support from the following observations.

(1) The part of the population wherein a preponderance of stable minds might be expected seems to suffer little from psychogenic disorders. This is noticeable amongst the higher grades of the Civil Service and is observable also amongst agricultural settlers from other colonies.

(2) Experience suggests that pure neuroses and psychoneuroses are not common amongst Kenya-born and reared youth.

(3) In the psychological treatment of settlers I have not yet failed to find that the neurotic disorder was imported.

(4) The physical health amongst the rural population after a variable period of residence is astonishingly good; the rural children are 'pictures of health.'

(5) A considerable proportion of imported psychogenic disorders obtains benefit and 'cure' without any treatment. It is permissible to ask, without discussing possible causes of this benefit, whether if light, heat, altitude and so forth were indeed the etiological villains of the piece, we should not expect deterioration rather than improvement to be the rule.

Undoubtedly some cases deteriorate; but in the tropics as elsewhere the neurologist who sees no clear dividing line between the organic and the functional, and recognises as yet no convincing separation of neurosis, psychoneurosis and psychosis from each other, allows himself the liberty of suspecting every case of neurosis. "Nerves from loneliness": "a touch of the sun now and then": "fear of driving a car and of talking to strangers": "bad memory and giddiness from the altitude": these were the complaints with which four recent settlers urged neurosis upon me. Under the first there was epilepsy from childhood; the second overlay alcoholic insanity with delusions; the third concealed early taboparesis; the last was that of a moral imbecile of wealthy family (pederastic with natives).

Without endowing them with causative value we may admit that the petty worries of pioneering and of isolated life may aggravate imported cases which do not benefit spontaneously from the climatic conditions and from the many new opportunities for sublimation of impulses, or which resist all available treatment We may see some aggravation also from lamentably unsuitable housing and daily habits, including those arising from the popular belief that whisky and cocktails are an urgent tropical necessity. But in the absence of stronger evidence than has come to my notice it may be reasonable to remain sceptical about the causative influence of light, heat, and altitude. In face of our knowledge of conflict, repression and complex, of dissociation and regression, we may think that this, small as it is, already enables us to ask for more restraint in the use of terms coined in former days to conceal ignorance, such as *tropical* memory and *tropical* temper. There lingers amongst practitioners in the tropics a tendency to discredit the fact that neuroses, psychoneuroses and some psychoses are psychogenetic in origin: not a few bristle with indignation at the suggestion that organic disorder may result from mental disorder, that each may aggravate the other. The white man in the tropics has an acquired fear of light and sunshine, bringing with it sometimes an unnecessary avoidance of the open air. There seems a little inconsistency somewhere when throughout a foggy November physicians in Harley Street recommend patients to that same light, sun and air. While we in Kenya may rejoice

at such sagacious advice, we are apt to wonder whether the modern specialist in heliotherapy will not contribute more of value than we can to a discussion of neuroses in the tropics.

As the situation appears to me, without the advantage of professional friction to expose the errors of my reasoning, it would be all to the good, medically and socially, if the white man could, on scientific grounds, be relieved of the fear already referred to. Whether or not this would be justifiable on the ground that tropical light and heat are to be regarded mostly as aggravating factors of existing psychogenic disorders—possibly from a power to induce nervous (synaptic) fatigue—may be a subject worthy of discussion. He might learn to advantage perhaps by analogy that when treated with proper consideration tropical heat is no more harmful than Alpine cold, while sunstroke and frost-bite are still penalties of folly. In this way also he might be persuaded that light and heat, enjoyed as advised, are even more beneficial than the dose taken as prescribed from the patent medicine bottle alarmingly labelled *poison*. A further and to my mind most important step towards a reasonable standard of life in the tropics should follow efforts to destroy faith in the tropical value of whisky and gin, and substitute reliance on the more engaging if less grossly dissociative effects of the excellent colonial wines and beers. Such a change might be painful to many; nevertheless it would be worth while because of the improvement that would ensue amongst our race, in Africa at least, in the functional activity of the synapses. I trust that it will offend no one if I add that small as these points may appear, there is a need for scientific dicta and stable reasoning to dispel dismay in tropical Africa caused by those who lament our white skin, prophesy early extinction of all xanthochroids, and bid us hasten to pigment ourselves by artifice.

My conclusions, then, are these:

(1) Neuroses in the tropics do not differ in nature and origin from neuroses elsewhere, or in the susceptibility of their organic accompaniments and results to aggravation by environmental influences.

(2) There appears to be no ground for the statements still met in the literature of tropical medicine that tropical light and heat have an inevitable injurious effect upon the nervous systems of our race.

(3) While the process we vaguely call *acclimatisation* is interpreted by some authorities as an acquiring of immunity to effects of tropical sun upon the nervous system, I have seen nothing of these inevitable deleterious effects and have not been fortunate enough to meet any scientific explanation of them. Research appears to be desirable. Meanwhile we have the belief (which my experience by eight years' residence in Southern Europe confirms) that the southern European has a naturally higher nervous susceptibility than the northern European, and one gropes in vain for explanation of the paradoxical view advanced in responsible quarters that in the tropics the southern

European's nervous susceptibility presents itself in guise more suitable to 'acclimatisation' than the naturally lower nervous susceptibility of the northern European.

(4) While physical factors cannot be disregarded in efforts to define 'acclimatisation' it might be well to examine also possible psychological factors. I venture the view that the process seems to be quick and effective in the extrovert, but is slow and may fail in the introvert. If this were confirmed it would attach to temperament at least as much importance as some now attach to colour of hair.

(5) The frequency of psychogenic disorders amongst whites in tropical countries cannot be considered rationally apart from careful estimation of the psychical as well as of the physical elements in the country's immigrants; this at present renders invalid any arguments favouring special tropical causes for these disorders. If the conclusion be correct (and experience in several tropical climates leaves me no doubt) that some of our tropical colonies receive too high a proportion of those who suffer from or are predisposed to psychogenic disorders, the fact may be connected with the old national belief, in family, school and office, that what may not be good enough for England will certainly be good enough for her dependencies.

(6) The question of susceptibility to neuroses, psychoneuroses and psychoses and that of mental stability generally, have social as well as medical and psychological aspects in a young colony, guidance on which by expert advice is increasingly desirable. In East Africa the problems are such that it is a matter of grave importance that colonisation should be carried out by our most stable-minded individuals. We are entitled to ask whether general, social and experimental psychology is in a position to assist towards this high end. At present the colonial statesman limits his interest in science to problems of research directed towards increase of profitable production. But we are assured by the statesman, to the conviction of all who have given thought to the subject, that the development of East Africa this century will be a test of our character and civilisation, perhaps a pivot of our destiny. It is, therefore, desirable to look beyond the question of profitable production to the question of profitable colonisation, and to ask whether the sciences we represent, with other allied sciences, do not owe a duty to the Empire—that of combining to establish *a science of colonisation* to displace our national habit of 'muddling through,' and to benefit immediately and remotely, socially and economically, the aims of Empire, as surely as public health, industrial psychology and scientific management in production benefit national aims.

Editorial.

THE TEACHING OF PSYCHOLOGICAL MEDICINE.

IT is more than a little surprising that despite the greatly increased interest in psychology that has developed in recent years, especially in the decade since the War, no corresponding change is evident in the curriculum laid down for the psychiatric training of medical students in this country. A perusal of the syllabuses of our medical schools shows that in most of them not only the courses prescribed, but the facilities offered, have increased but slightly in the last twenty years. There is still in most schools the brief course of a dozen or so systematic lectures, with a similar number of visits to an asylum for the purpose of clinical instruction; but there is seldom any local ordinance (as there is no general one) compelling more than that. And the conditions under which the clinical teaching is done are not favourable; for in psychiatric clinical teaching alone is it the custom to have a class so unwieldy in numbers that to teach the individual student is an impossibility. The so-called clinical work seldom consists in more than a lecture at which some patients are presented. The general effect on the students is foreign to the spirit of medicine, and is more akin to the feeling induced by witnessing some curious spectacle.

Further, these courses, inadequate as they are (from no fault of the individual teacher) are given in the middle or towards the end of the clinical period. This is in spite of the fact that in the wards of any general hospital can be found psychological material, chiefly of the psychoneurotic, but often of the psychotic kind; and in disregard also of the seldom recognised but important circumstance that a large percentage of the patients in private practice consult their doctor for symptoms that have only the strong semblance of a physical origin. For the doctor whose eyes have been opened in this direction (and he is still a rare bird, for, as we have said, the subject cannot be held to be adequately taught in the schools) the percentage of such patients in his practice is high indeed. But more usually one hears the general practitioner remark in a half-apologetic way, "I see only one or two cases of this kind in a year." All

the while the unqualified practitioner and the qualified man who can blind himself so far as to be enthusiastic in the use of some new-fangled panacea, are producing undoubted "cures," where the more orthodox practitioner has failed; and the forms of religion which lay their emphasis on bodily healing increase their adherents many-fold. Perhaps Medicine would have no ground for taking exception to this state of affairs, if such cures by such methods were the soundest that could by any means be obtained. But it is a first principle of medical practice to treat the disease rather than the symptoms; and there is little doubt that the practitioners and the cults alluded to treat merely the symptoms. Further, the spirit that inspires the progress of medicine works towards the increase of understanding; and these cults and practitioners do not trouble deeply to understand.

It is therefore a pity that when there exists a body of knowledge that is now as complete as that in most branches of medicine, it is not made available in the schools; for the courses in vogue there do not (as a rule) make it available. The student who is required to study pathology is first of all grounded in normal histology; before he begins his bedside work he is taught physiology; but he is introduced to the clinical study of mental disorders (in most instances) before he knows any more of the mind than he might have picked up in desultory reading, or by commonsense. Nearly half of our medical schools offer preclinical courses in normal psychology; but only two or three of these make the course compulsory. Two or three schools at most offer any instruction in abnormal psychology. The case is far otherwise in the Dominions, where nearly every school of standing has a compulsory course of preclinical psychological work; and where frequently the prescribed clinical instruction is much more nearly adequate than it is with us. One Dominion university goes so far as to make psychiatry one of the four principal subjects of the final examination. In America, in the leading schools there, the medical student is introduced to the subject of psychology in his first year, and by means of lectures, demonstrations and individual work with patients, is never permitted to lose contact with the psychological aspects of medicine throughout all the years of his curriculum. Compared with these arrangements, what is provided in medical schools in this country (with a few conspicuous exceptions) is meagre indeed.

It is still a foremost principle of British Medicine to treat the patient as well as the disease. In so far as the practitioner

obeys this principle, he is consciously or unconsciously using psychological methods. This is as it should be; but is it a departure from tradition to formulate the treatment on considered and established rules, with the fullest insight that scientific observation can give, rather than by rule of thumb?

If we were asked to formulate a scheme of psychiatric instruction, which, without being cumbrous and making a disproportionate demand on the already too much occupied medical curriculum, would yet furnish a satisfactory basis on which to construct experience, we would suggest something like the following.

A course of lectures on normal psychology (with some simple experimental work) in the same period as physiology is being studied, should be followed in the next year by a course of lectures on the principles of abnormal psychology, embellished wherever possible with clinical illustrations. The abnormal psychology so taught should be chiefly of the kind most likely to be useful to the student; i.e., the types expounded by Dejerine, Meyer, and in this country by Ross. (The more recondite and less immediately useful parts of the Freudian psychology could safely be left to the specialists, or to individual curiosity). It is desirable that teaching in abnormal psychology should not be deferred till the late clinical period. It should form a groundwork for the student who proceeds in one or other of his last two years to a clinical clerkship in an out-patient psychiatric department. The present statutory course in the principles and practice of psychological medicine would stand most conveniently in the fourth year, and according to the exigencies of the curriculum, clinical instruction at a recognised mental hospital could be given in this or in the final year. The latter should preferably be taken in the year in which the student is not acting as clinical clerk in an out-patient psychiatric department, thus ensuring that his clinical psychiatric interests shall be spread out over most of his general clinical period. It is at present Utopian to conceive the substitution for the usual clinical demonstration at a mental hospital, of clinical clerkships with the same type of material; for this will be feasible only when psychiatric beds are provided in general hospitals, or when psychiatric clinics, such as nearly every university town in Germany possesses, are provided throughout this country.

Abstracts.

Neurology.

NEURO-ANATOMY AND NEUROPHYSIOLOGY.

[1] **The structure and connexions of the dentate nuclei of the cerebellum** (Structure et connexions des noyaux dentelés du cervelet).—V. Demole. *Arch. Suisses de Neurol. et de Psychiat.*, 1927, xx, 271.

A long, elaborate, and finely illustrated study of the dentate nuclei in health and disease enables the author to draw the following conclusions, among others.

Structure.—The dentates are formed of two distinct parts, dorsomedian and lateroventral respectively. These differ in cells, fibres, and general dimensions.

Direct Connexions.—The dorsomedian part (microgyric, palæocerebellar) receives fibres from the dorsomedian section of the cerebellar cortex—the palæocerebellum; the lateroventral (macrogyric, neocerebellar), from the cortex of the lateral lobe—the neocerebellum.

Indirect Connexions.—Each dentate is in communication with the opposite cerebral hemisphere; the dorsomedian region is in relation with the rolandic sensorimotor area (palæocerebral), the lateroventral with the sensory temporo-parietal area (neocerebral). Because of the size of its cells and its connexions with the rolandic region the dorsomedian part of the dentate may be regarded as possessing some kind of motor function.

Crossed cerebellar atrophies.—When crossed cerebellar atrophy follows hemispheric lesions it predominates in the lateral lobe; nuclear changes affect mainly the dentates and the nucleus emboliformis, whereas the n. globosus and roof nuclei are practically intact. Secondary atrophy of the dentate precedes that of the cerebellar cortex and can be detected two to three years after certain lesions of the internal capsule. The route of the atrophy is direct, via the pons and middle cerebellar peduncle, and retrograde, via the thalamus, nucleus ruber, and superior cerebellar peduncle. In longstanding cases the atrophy is not confined strictly to the appropriate cerebellar lobe; it may cross the middle line and invade the other lobe, even reaching the inferior cerebellar peduncle and spinocerebellar fibre-systems. It is different from Wallerian degeneration in that it slowly involves linked series of neurones, and is progressive; further, it has a profound affect on structure and function, and throws light on some cases of chronic nervous diseases in which the symptoms become progressively worse.

S. A. K. W.

[2] **The role of the sympathetic nervous system in muscle tonus.**—S. W. Ranson and J. C. Hinsey. *Jour. of Comp. Neurol.*, 1926, xlii, 69.

A series of ten cats were sympathectomized, the left lumbar sympathetic cord being removed through a median abdominal incision. From 50 to 152 days intervened between sympathectomy and decerebration. Observations made before and after decerebration showed no constant variation between the hind limbs. Maintenance of extensor rigidity, presence of lengthening and shortening reactions, and resistance to passive flexion were equal in the normal and the sympathectomized hind limbs.

The conclusion is drawn that the sympathetic nervous system is not responsible for the exaggerated tonus of decerebrate rigidity.

In three of the sympathectomized cats tetanus toxin was injected in equal amounts subcutaneously over each femoral trochanter. Observations recorded from day to day did not show any constant variation in the posture of the two sides or in resistance to passive movement. The action of the toxin was apparently alike on the two sides.

The conclusion is reached that the exaggerated tonus caused by the injection of tetanus toxin is due neither to an action of the toxin on sympathetic endings in the muscle nor to its producing an increase in tonic discharges from the spinal cord by way of the sympathetic system.

In another series of twelve cats sympathectomized on one side observations were made at intervals of two days for two weeks following the operation. The authors were unable to observe any constant variation in posture on the sympathectomized side in standing, running, supporting the body weight on the hind limbs, or jumping from a table to the floor.

S. A. K. W.

[3] **Electromyographic studies in different types of neuromuscular disturbances.**—C. P. Richter and F. R. Ford. *Arch. of Neurol. and Psychiat.*, 1928, xix, 660.

Electromyograms from voluntary contractions in syringomyelia, progressive muscular atrophy and poliomyelitis are all similar, and all deviate widely from the normal. The authors suggest that these peculiar currents are characteristic for lesions of the anterior horn cells, common to these three different diseases.

The records from patients with muscular dystrophy also deviate from the normal, but in a different way. The primary waves are much more regular both in frequency and in amplitude than is usual in normal persons, and they are almost entirely free from all irregularities. The conclusion is reached that the primary waves in the myograms from the three patients with lesions of the anterior horn cells are produced by the proprioceptive impulses. On the other hand the records from patients with muscular dystrophy lend support

to the view of Weizsäcker and Dusser de Barenne, who arrived at the conclusion that the primary waves are somehow produced by an inherent or autochthonous rhythmic activity of the motor horn cells.

R. M. S.

[4] **The part played by the supravestibular connections in decerebrate rigidity.**—L. J. J. Muskens. *Jour. of Physiol.*, 1928, lxiv, 808.

In former publications on decerebrate rigidity writers have dealt with the subject primarily as physiologists, anatomical considerations coming second.

Forced movements backwards in the vertical plane, whether caused by a lesion of the primary vestibular centre, by section of ascending secondary connections, by lesion of the supranuclear centre near the posterior commissure or its palæostriatal connections, are probably always associated with a certain amount of rigidity, recalling Sherrington's "composite postural reflex, the antigravity muscles counteracting the superincumbent weight."

From different observations it is seen that there is a complete reversal of physiological effect at the level of the posterior commissure. (1) In a series of oral to caudal hemisections across the brainstem of a quadruped, temporary rigidity, homo- and hetero-lateral, is noted. (2) If an incision is made in the region of the posterior longitudinal bundle forced movements in the frontal and horizontal planes are directed reversely, according as the incision is on the caudal or the oral side of the commissure. (3) The same complete reversal is observed after faradic stimulation of the same region, when oral-caudal sections are made through the brain of primates.

Since section of the pyramidal tract has probably nothing to do with the origin of decerebrate rigidity, and as the nucleus ruber is frequently found intact, the part played by supravestibular connections in the production of the phenomenon may be supposed to be considerable. This applies especially to the ascending and descending tracts, lesion of which is associated with forced movements in the vertical plane.

S. A. K. W.

NEUROPATHOLOGY.

[5] **Cerebral softening, its frequency, its site, and the state of the arteries in the affected region** (Contribution à l'étude du ramollissement cérébral envisagé au point de vue de sa fréquence, de son siège et de l'état anatomique des artères du territoire nécrosé).—Foix and Ley. *Jour. de Neur. et de Psych.*, 1927, xxvii, 658.

This article was in preparation by the authors jointly at the time of Foix's death, and has been finished by the pupil, following as far as possible the former's ideas. Softening is considered to be synonymous with arterial blockage by thrombosis or embolism.

1. *Relative frequency of softening and hæmorrhage.*—Neglecting small lesions, both thrombotic and hæmorrhagic, the examination of the brain in 124 cases of localised vascular lesions in elderly people showed that there were 100 cases (80 per cent.) of softening, and 24 (20 per cent.) of hæmorrhage. An engorged softening is probably often mistaken at post-mortem examination for hæmorrhage. It is certain that the great majority of hemiplegics who survive the ictus are cases of thrombosis; hæmorrhage is much more often primarily fatal. Even allowing for this fact softening is the more frequent. Among 66 cases of stroke fatal within three weeks, there were 47 (71 per cent.) of softening and 19 (29 per cent.) of hæmorrhage. Among 60 cases of long-standing hemiplegia there were 53 (88 per cent.) of softening and only 7 (11 per cent.) of hæmorrhage.

As regards the prognosis—neglecting the very mild ones—of 24 cases of hæmorrhage 17 (70 per cent.) were fatal within three weeks; among 100 cases of softening 47 (47 per cent.) were recent cases and 53 old. In the average of all the cases 64 were recent and 60 old, i.e., a mortality (within three weeks of the stroke) of 51·6 per cent.

2. *Site of the softening.*—In these 100 cases of softening the authors found the lesion 69 times in the distribution of the middle cerebral artery, 19 times in that of the posterior cerebral, and 12 times in that of the anterior. In the 69 cases of middle cerebral artery blockage, the superficial distribution was affected alone in 50·7 per cent., the deep distribution alone in 29 per cent., while 20·3 per cent. were mixed cases.

3. *The state of the arteries in the softening.*—The arteries concerned were minutely examined in 63 cases of softening. In 19 cases (30 per cent.) the lumen was obliterated, and in 7 of these the obliteration was due to embolism. There were 14 cases (22 per cent.) in which there was "sub-total obliteration," i.e., the lumen had become filiform, but still macroscopically patent; there were 30 cases (48 per cent.) in which there was incomplete blocking and in three of these the artery appeared almost healthy, but was smaller than normal. It will be seen that complete blockage is far from being the rule (only 30 per cent. of cases).

In the great majority all the cerebral arteries were unhealthy, and the softening had not by any means always occurred in the territory of the most affected artery. Microscopic examination in 26 cases showed the usual characters of chronic arteritis.

There are three conditions which may give rise to softening; in the 63 cases minutely examined the findings were:—

1.	Embolism	7 cases (11 per cent.).
2.	Arteritis	53 cases (84 per cent.).
3.	No appreciable disease	3 cases (4·7 per cent.).

If the cases of arteritis are considered alone it is seen that in more than half of them the artery involved is quite permeable; furthermore the disease is chronic and the state of the arteries cannot be suddenly worse on the day

of the stroke than on preceding days. There must consequently be a precipitating cause of the softening. The authors think that in most cases a relatively acute ischæmia is probably brought about by spasm of a diseased artery, and in some others by general cardiovascular insufficiency, while in a few a toxic state of the blood (uræmia) may be a factor in causing the softening. These three factors should be considered in the treatment of a stroke during the acute stage.

J. P. MARTIN.

[6] **Senile plaques** (Étude histo-chimique des plaques séniles).—DIVRY. *Jour. de Neur. et de Psych.*, 1927, xxvii, 643.

THE optical properties, staining affinities and solvents of *plaques séniles* show that they are hyaline and closely related to amyloid substance. They are the expression of a miliary disseminated amyloid process affecting particularly the cerebral cortex.

J. P. M.

[7] **Localisation in animals of streptococci from cases of epidemic hiccup, encephalitis, spasmodic torticollis and chorea.**—E. C. ROSENOW. *Arch. of Neurol. and Psychiat.*, 1928, xix, 424.

THE author reports a further series of experiments on animals, performed with material from foci of infection or throats of patients suffering from various diseases of the nervous system. The results corroborate and extend the former observations.

The streptococci isolated in each of these conditions, while much alike, possess very different localising and symptom-producing power when injected intracerebrally into animals and readily lose this property, especially on aerobic cultivation. The specific localising power of the streptococcus, it would seem, is dependent, in part at least, on the production of a toxin or poison that has specific effects. Lethargy, hyperpnœa, with marked arrhythmia, and the symptoms characteristic of spasmodic torticollis have been produced with filtrates from nasopharyngeal washings, filtrates of suspensions of pus from tonsils, and filtrates of old cultures of the specific streptococcus in each of these conditions. The fact that the streptococcus, having specific effects, and the power to produce a specific poison, has been isolated long after onset indicates that the late manifestations of encephalitis and allied conditions are not sequels but are due to the activity of the causative organism or its toxin, as has been especially emphasized by Freeman.

It is of course realised that the results obtained are not absolute proof of the etiological relationship of the streptococcus isolated. None of the patients in this group died, hence search for the organism could not be made. Agglutination experiments, especially with the serum of patients and the suspension of the suspected organism, while sometimes positive in low dilutions, were not so regularly enough to be convincing. The benefits which follow the removal

of foci proved to contain the organism and the use of autogenous vaccines, while often striking, do not occur regularly enough to be of specific etiological value.

In consideration of all the facts, especially the extremely specific effects obtained in some cases with the streptococcus isolated, and its specific toxin- or poison-producing power, the tentative conclusion seems warranted that this organism is of etiological significance in each of the diseases studied.

R. M. S.

[8] **The perivascular pia-glia membrane of the brain** (Die perivasculäre Piagliamembran des Gehirns).—G. SCHALTENBRAND and P. BAILEY. *Jour. f. Psychol. u. Neurol.*, 1928, XXXV, 199.

THIS is a very fine piece of histological research, illustrated by over 80 reproductions of microscopical preparations. Only the conclusions of a long paper can here be summarised.

1. The perivascular reticular connective tissue, the macroglia, and the pia-glia membrane formed by these two, tend to hyperplasia in numerous degenerative and inflammatory conditions of the central nervous system.

2. The perivascular reticular tissue, in such circumstances, is responsible for the appearance of fibroblasts, epitheloid cells, and probably also of phagocytes (Körnchenzellen and macrophages).

3. The lymphocytes, plasma-cells, and polymorphonuclear leucocytes of "inflammatory" states find in the pia-glia sheath a check to their invasion of the neural parenchyma.

4. In cases where these hæmatogenous elements reach the parenchyma it can be shown that the macroglia cells have undergone pathological change in the form of swelling, vacuolation, loss of processes, failure of fibre-formation, and regressive alteration of nuclear tissue.

5. In degenerative states of neural parenchyma the perivascular pia-glia membrane allows the Körnchenzellen derived from microglia to pass in the direction of blood vessels. In addition to direct diapedesis there would appear to be some enclosing of the arriving Körnchenzellen in mesenchymal tissue.

6. Piogenic tumours (meningiomas) respect the pia-glia membrane longer than they do both dura and bone. If sarcomatous, the membrane is broken through.

7. Gliomata usually effect hyperplasia of the membrane, the exception being highly fibrillar gliomata such as unipolar spongioblastoma and fibrillar astrocytoma.

8. As long as glioma cells can produce real glia fibrils and "vessel feet," so long does the connective tissue remain confined to the vascular system; when the former fails, then an entangled connective-tissue network or stroma develops within the tumour.

9. Vessels within gliomata and pial tumours tend to degenerate.

S. A. K. W.

[9] **Testicular lesions in rabbits following int.aspinal syphilitic infection.**—G. W. Raiziss and M. Severac. *Proc. Soc. Exp. Biol. and Med.*, 1927, xxiv, 756.

The investigators studied the systemic manifestations of syphilis in rabbits infected intraspinally with spirochætes. This resulted in the striking observation that the majority of animals develop syphilitic manifestations when infected by the spinal route only. Of even greater interest is the fact that practically all of the rabbits exhibiting systemic infections developed testicular lesions.

Thus in one instance the animal exhibited a node in the left testis 53 days after being inoculated intraspinally with spirochætes; in other cases the latent period was longer, up to 187 days. Examination showed that the nodes contained actively motile spirochætes. Previous investigations (Brown and Pearce) indicate that spirochætes migrate from the site of testicular inoculation into the cerebrospinal system; the present series of experiments prove that they travel also in the opposite direction. S. A. K. W.

[10] **Resorption of cerebrospinal fluid through the choroid plexus.**—H. S. Forbes, F. Fremont-Smith and H. G. Wolff. *Arch. of Neurol. and Psychiat.*, 1928, xix, 73.

By the method of introducing isotonic potassium ferrocyanide and iron ammonium citrate solution into the subarachnoid space under pressures closely approaching the normal, Weed showed that the chief channels of escape of the cerebrospinal fluid were the arachnoid villi. If, however, a hypertonic solution of sodium chloride were previous injected into a vein, accessory paths of elimination were ôpened, probably due to a reversal in direction of flow. These observations were substantiated by Foley, who in addition showed the presence of Prussian blue granules within the vessels of the choroid plexus. The present paper deals with the results of experiments designed to demonstrate Prussian blue granules in the same situation after intravenous or intraperitoneal injections of hypertonic salt solution, while maintaining intracranial pressure within physiological limits. In each of the twelve animals examined Prussian blue granules were found within the vessels of the choroid plexus. This would indicate that the direction of flow through the choroid plexus may be reversed by increasing the osmotic pressure of the blood, which in its turn suggests that the choroid plexus is not a secreting gland but a semipermeable membrane through which the direction of fluid exchange is determined largely by the balance between hydrostatic and osmotic forces. R. M. S.

[11] **The Kafka paraffin reaction in the spinal fluid.**—L. J. Karnosh and A. J. Rademaekers. *Arch. of Neurol. and Psychiat.*, 1928, xix, 125.

The authors applied this test to the spinal fluid of ninety-one patients, and find that it compares favourably with the gum mastic reaction.

R. M. S.

SENSORIMOTOR NEUROLOGY.

[12] **Mindblindness** (Zur Klinik der Seelenblindheit).—P. SCHUSTER and H. TATERKA. *Deut. Zeits. f. Nervenheilk.*, 1928, cii, 112.

A DESCRIPTION is given of five personal cases in which the prominent clinical feature was so-called mindblindness, a variety of agnosia. In four of the five definite hemianopia was present; in the fifth, it was probable. Acuity of vision was unimpaired. Disorientation in space was usually more or less pronounced, and, as in common enough, was more noticeable when the patient was being tested than in his ordinary spontaneous activities. As for the mindblindness (visual agnosia) it showed itself in the failure of the patients to recognise and use the ordinary objects of daily service (watch, spoon, glove, etc.) and in a similar failure as far as their pictorial representation was concerned. More elaborate pictures were of course even more confusing. One patient invariably failed to recognise the physician. Yet, again as usual, visual perception was not at fault. The visual qualities of a watch, its shape, colour, metallic nature, polished appearance, etc., were always visually appreciated. In contrast to some other recorded cases of the same condition, the authors' patients as a rule were able to give sufficiently complete visual descriptions of the features of a given object named to them, such as a tuning fork, and it was remarkable that the patient should absolutely fail to recognise a real tuning fork handed to him when a moment before he had described it fully from memory. On some occasions, again, optical qualities were attributed to objects in description which as a fact they do not possess—a kind of visual confabulation.

In all the cases, in greater or less degree, tactile agnosia (inability to name an object by touch) was observed, and this has been a feature of other recorded cases; it too, was a phenomenon noticed in more striking fashion during examination than when the patient was left to himself. The stereognostic qualities of the test objects were usually recognised. An interesting observation was made in some instances, viz. that the patient recognised the object better by the tactile route when the eyes were shut. Motor apraxia was not noted, whereas alexia was for obvious reasons prominent.

Subsequent examination showed that in all five cases the lesions were approximately in the same region, viz., left cuneus, calcarine area, gyrus fusiformis, gyrus lingualis.

S. A. K. W.

[13] **Mental symptoms and post-mortem appearances in Huntington's chorea.**—W. A. T. LIND. *Med. Jour. Austral.*, 1927, xiv, 53.

THE author publishes the clinical histories and post-mortem findings in six cases of Huntington's chorea. He regards the insanity in this disease as one of the dangerous type, its most marked features being sudden outbursts of violence, sexual excesses and a tendency to suicide.

Clinically he views the peculiarities of speech, posture and gait as being dependent on the jerkiness of the chorea. A true Rombergism never exists.

His notes on the post-mortem findings in the central nervous system itself are scanty, but he states that, contrary to the usually accepted view, none of his cases showed any evidence of meningoencephalitis. In only one case was there the slightest thickening of the pia-arachnoid. The most marked finding was the extreme and usually general convolutional atrophy. There was no involvement of ependyma or glial reaction. In five out of his six cases there were no signs of cerebral arteriosclerosis.

In all the wasting of the brain had been so gross as to destroy the basal ganglion pattern.

Microscopically, in one case he found evidence throughout the brain and cord of cell degeneration and minute hæmorrhages in all areas; in another of cell degeneration and vascular engorgement without hæmorrhage. He suggests that the meningeal changes recorded at autopsy in cases of Huntington's chorea have been due to co-existent pathological conditions, and points out that a disease so markedly hereditary is not likely to be a meningoencephalitis.

P. W.

[14] **Cerebral tumours without papilloedema** (Tumeurs cérébrales sans papille de stase).—MARTIN and VAN BOGAERT. *Jour. de Neur. et de Psych.*, 1927, xxvii, 756.

EIGHT cases are described in which tumours were present in widely different parts of the brain, papilloedema being absent until a late stage. The authors recommend Baillart's plan of measuring the pressure in the central artery of the retina when increase of the intracranial pressure is suspected.

J. P. M.

[15] **Cholesteatoma of the brain.**—F. A. KAY and G. T. PACK. *Arch. of Neurol. and Psychiat.*, 1928, xix, 446.

A REPORT is given of a non-hair-containing, epidermoid cholesteatoma, unique because of its unusual size. The tumour weighed 69.5 grm. and measured 4 by 5·5 by 8 cm. Its exterior had the lustre of mother-of-pearl and was composed of multiple nodules, each of which was made up of laminated waxy material. These laminæ were undoubtedly layers of flattened cornified squamous cells, somewhat resembling those of the epidermis. The tumour originated from the pia mater at the cerebellopontine junction and flattened the cerebellum, pons and cerebral peduncles.

R. M. S.

[16] **Torula histolytica meningoencephalitis.**—G. W. HALL, E. F. HIRSCH and H. MOCK. *Arch. of Neurol. and Psychiat.*, 1928, xix, 689.

THE authors report a case of infection of the brain and meninges by *Torula histolytica*, a yeast-like organism which reproduces by budding in tissues and in cultures, but which differs from the true yeasts by not fermenting sugars.

The course of the illness was marked by an insidious onset, persistent severe headache, and slowly developed signs suggestive of brain abscess, some form of meningitis or possibly brain tumour. The diagnosis was established by culture of the cerebrospinal fluid.

How the organisms reached the meninges could not be determined; possibly the infection came through the nasopharynx.

R. M. S.

[17] **Epidemic encephalitis.**—T. P. Sprunt. *Amer. Jour. Med. Sci.*, 1927, clxxiv, 660.

The author analyses a series of fifty cases showing chronic manifestations. He found no evidence of constitutional or hereditary predisposition. Thirty-two of his cases gave a history of having had "influenza," that is, some illness in which fever, diplopia, somnolence, insomnia, delirium and ptosis were usually symptoms.

Motor disturbance predominated greatly over sensory. The only cases showing obvious autonomic disturbance were those with marked sialorrhœa. In most of the cases the cerebrospinal fluid showed no change nor was the B.M.R. altered.

Mydriasis was noted in 19 cases, myosis in one, and anisocoria in eight. In ten the pupils reacted "sluggishly" to light, and in one they were fixed. No other eye signs are commented on, nor is any note made of the reaction on convergence.

From a prognostic point of view he considers the cases with the more violent onset to be more liable to develop ultimate Parkinsonism. Hyoscine in doses of 1/75 gr. t.d.s. is the drug of greatest value in treatment.

P. W.

[18] **Paroxysmal oculogyric crises in Parkinsonian encephalitis.**—Irving Pardee. *Amer. Jour. Med. Sci.*, 1928, clxxv, 683.

Pardee describes four cases of Parkinsonism which showed oculogyric crises. In all the conjugate deviation of the eyes was to the left and upward. It was usually associated with a cephalogyric torsion movement of the head to the left. The crises in most cases appear two or more years after the acute attack.

In all cases there are associated hyperkinetic phenomena—torsion of head and neck, respiratory episodes, tongue spasms and so on. Other accompanying phenomena may be emotional outbursts, head pain, partial loss of consciousness and vegetative symptoms.

Pardee stresses the paroxysmal character of the crises. He concludes that the hyperkinesis of which this is a manifestation is produced by a subcortical, striatal, extrapyramidal mechanism. The conjugate deviation is dependent upon a posterior longitudinal bundle-anterior corpus quadrigeminum complex; the hypertonic crises together with other torsion phenomena bring these two mechanisms together, and point to a striatal-anterior

corpus quadrigeminum-posterior longitudinal bundle mechanism. Etiologically the condition is one of the later manifestations of post-encephalitis lethargica.

P. W.

[19] **The labyrinthine functions in oculogyric crises** (Recherches sur l'état des fonctions vestibulaires dans les crises oculogyres de l'encéphalite). —Helsmoortel and van Bogaert. *Jour. de Neur. et de Psych.*, 1927, xxvii, 574.

Ten cases were investigated, and in most of them some slight hypo-excitability of labyrinthine functions was found. This is attributed to lesions of the vestibular nuclei and of the globus pallidus. The authors consider that the cause of oculogyric crises is possibly a transitory hyperæmia in the globus pallidus. J. P. M.

[20] **Postencephalitic supranuclear vestibular disorders** (Les troubles vestibulaires supra-nucléaires postencéphalitiques).—L. J. J. Muskens. *Revue neurol.*, 1927, ii, 155.

The phenomena of forced movements readily obtainable in the experimental animal are practically never found in man. Unilateral section of a posterior longitudinal bundle invariably results in circus movements on the sound side and rolling movements to the affected side. The direction of these two forced movements is reversed if the posterior commissure is implicated. Evidence suggests that lesions of the nucleus of the posterior commissure are responsible for the circus movements while lesions of the nucleus interstitialis account for rolling movements. Fibres fine in calibre, descending from the posterior commissure, send collaterals to the third and sixth nuclei, and coarser fibres of the interstitiospinal tract join the fourth. With involvement of these collaterals the phenomenon of skew deviation may be associated. The globus pallidus is in relation to the region of the posterior commissure, but lesions at levels between the two do not appear to alter the direction of the forced movements obtained as is mentioned above. It is probable the connection of globus pallidus and commissural nuclei is double, so that reported cases of conjugate deviation of eyes and head in striatal lesions may thus be explained.

Certain symptoms of postencephalitic Parkinsonism deserve consideration along the same lines. Paralysis of conjugate deviation occurs with some frequency, and paralysis of vertical movement also; the author states that the "anatomical localisation of these lesions is unknown," which is a somewhat misleading comment, and evidently favours the view that supravestibular connections are involved.

A discussion of oculogyric crises follows. In the author's opinion "lesions corresponding to oculogyric crises should be found in the regio subthalamica or locus niger in front of the nuclei of the commissure." In this region the fibre-systems lesions of which cause forced movements in the vertical or horizontal

plane are sufficiently close to each other. Spasmodic movements of eyes in an upward or lateral direction are said to be of supranuclear vestibular origin.

S. A. K. W.

[21] **Spinal and peripheral forms of epidemic encephalitis.**—(Sur les formes basses de l'encéphalite épidémique).—LEY and VAN BOGAERT. *Jour. de Neur. et de Psych.*, 1928, xxviii, 19.

AT the beginning of this paper five cases are described which presented more or less vague symptoms suggestive of inflammation of the spinal cord or peripheral nerves; in four of them there was also slight diplopia; all ended in recovery within a few weeks. In none of them was there any prolonged sleep nor is any satisfactory evidence given that the illness in any resulted from the infection of epidemic encephalitis, as the authors guardedly suggest.

In the discussion of the diagnosis of these cases three other cases are described which might equally well have been regarded as resulting from the same infection. One of them subsequently turned out to be a case of disseminated sclerosis, while in another no diagnosis was arrived at, even post-mortem.

J. P. M.

[22] **The thalamic and Parkinsonian types of infundibular tumours.**—L. VAN BOGAERT. *Arch. of Neurol. and Psychiat.*, 1928. xix, 377.

Two cases of infundibular tumour are described.

The first gave rise to an adiposo-genital syndrome with disturbance of glycoregulation; this was soon complicated by a thalamic syndrome and later by a Parkinsonian syndrome. Anatomical study revealed a large tumour of the adamantinoma type which had apparently developed at the expense of the original superior epithelial cells of the infundibulum; from there it had invaded the tuberian floor, filled the cavity of the third ventricle and pushed the thalamic masses aside laterally; it had invaded the lateral ventricles, which it distended greatly, and had pushed aside the white substance of the frontal pole and the central gray nuclei. Histologically, the globus pallidus showed marked lesions; the pituitary gland was intact.

In the second case, the development of a syndrome of intracranial hypertension led rapidly to a thalamo-infundibular syndrome. The infundibular symptoms were polyuria, disorder of glycoregulation and crises of narcolepsy with onirism, fabulation and retrograde amnesia. The thalamic symptoms were pains, hyperæsthesia to cold and trophic changes in the extremities. The extrapyramidal syndrome was represented by slowness, retropulsion and tremor. X-ray examination confirmed the existence of a tumour above the pituitary gland.

The author concludes that the thalamic syndrome must be referred to destruction of the internal nucleus of the thalamus; the true Parkinsonian

syndrome, to a lesion of the globus pallidus. Some extrapyramidal disturbance must be attributed to functional troubles in the frontal pole as the result of dilatation of the lateral ventricles from neoplastic extension.

During the infundibular phase glycoregulation is, as a rule, disturbed, and may be diminished. The meaning of this is not established. The endocrine disturbances are almost always characterised by an adiposogenital dystrophy which may later evolve into a marked cachexia.

In the first case the histopathological integrity of the pituitary gland is of biological interest in relation to the disturbance of glycoregulation, the disorder of fat metabolism and the regulation of sexual function.

R. M. S.

[23] **An analysis of abnormal posture of the head in Parkinsonism of various origins.**—W. M. Kraus and N. E. Silverman. *Arch. of Neurol. and Psychiat.*, 1928, xix, 301.

In 1927 Kraus introduced the term "dissolution of erectness" to describe the posture in Parkinsonism, which is usually a combination of mask-like facies, forward flexion of the head and trunk, flexion at the hips and knees, adduction of the arms and flexion of the elbows and fingers. This classic dissolution of erectness does not, however, always occur in all details, and in the present paper the authors have analysed the various atypical positions of the head which may be encountered. For this purpose lateral views of patients with Parkinsonism were carefully traced from photographs, and two lines were drawn, one from the external meatus to the outer canthus, another from the external meatus through the trunk and crossing a horizontal line corresponding to the floor. The angle made by the meatus-canthus line with the meatus-floor line is called the head-trunk angle. The angle made by the meatus floor-line with the horizontal floor-line is called the trunk-base angle. The normal head-trunk angle is 110 degrees; the normal trunk-base angle is 90 degrees.

In a group of seventeen cases of Parkinsonism the trunk-base angle was always less than 90 degrees, irrespective of the position of the head. The head-trunk angle varied from the normal in either direction; to 154 degrees in extension, and to 71 degrees in flexion.

In addition to the dorsal and ventral flexion of the head, there is, as a rule, lateral flexion and rotation. Thus the flexion forward of the head, such as occurs in pure dissolution of erectness, is obscured by these deviations in all three planes. An increase of the head-trunk angle has not been found in the degenerative types of cases. It is common in the encephalitic type.

R. M. S

[24] **Tumour of the third ventricle with Parkinsonian and thalamic syndromes and disorders of sugar metabolism** (Tumeur du troisième ventricule avec syndrome parkinsonien et thalamique ; troubles de la glycorégulation ; étude anatomique).—Van Bogaert, Nyssen and Ley. *Jour. de Neur. et de Psych.*, 1927, xxvii, 779.

This is the record of a remarkable case. The first symptoms were frequent epileptiform attacks, and soon after the onset of these the patient began to increase rapidly in weight. Eighteen months later he was found to have hemianopia (he had only one eye), and a commencing atrophy of the optic disc. He had become sexually impotent. Sugar tolerance was greatly increased. In the next few months he developed intense pain in the left half of the body, and hyperalgesia, and became blind. Two years after the onset a Parkinsonian syndrome came on and the patient began to suffer from cachexia. Finally he had paralysis in flexion.

At autopsy a large suprahypophyseal tumour was found which seemed to have grown from the infundibulum and spread thence along the floor of the tuber cinereum, filling the third ventricles, pushing the thalami laterally ; it had invaded the lateral ventricles and caused great enlargement of them, damaging inter alia the globus pallidus. The tumour resembled the hypophyseal adamantinomata but was of mixed structure. The pituitary was histologically intact. J. P. M.

[25] **A brief discussion of the thalamic syndrome with a case report.**—E. S. Gurdjian. *Amer. Jour. Med. Sci.*, 1928, clxxv, 18.

The writer reports a case of this syndrome occurring in a young married woman of 34. The first symptoms which appeared five days after a miscarriage were a peculiar feeling down the whole right side of the body, and inability to speak for two hours. Ten days later she noticed that the right leg dragged in walking. This lasted for a year, during which time the whole right side felt "as though asleep." There was no hemiataxy or hemiathetosis, but spontaneous paroxysmal pains occurred on the right side.

There was some impairment of stereognosis in the right hand, and slight loss of postural sense. Deep pain and vibration sense were considerably diminished in the right hand. Light touch was felt as a painful stimulus all over the right side. There was no sphincter disturbance. She had a high blood pressure but no signs of renal disease. The blood Wassermann reaction was negative.

The author comments on the rarity of the syndrome as early as 34, and explains this on the ground that vascular changes in the thalamus are its commonest cause. He suggests an embolus following on uterine thrombosis as the cause in his case. The associated signs of involvement of surrounding structures which are almost always found, and are probably due to œdema, were absent in this case. Spontaneous pain he considers due to pressure caused by gliosis of the infarcted part on incoming thalamic fibres. P. W.

[26] **Postural variations in nystagmus** (Quelques mots au sujet du nystagmus de position).—Buys and Hennebert. *Jour. de Neur. et de Psych.*, 1928, xxviii, 147.

In 1913 Bárány described two cases in which nystagmus was induced or varied according to the position of the patient's head in space. The first was that of a woman with disseminated sclerosis in whom a horizontal nystagmus towards the left was present as long as she lay with the right side of her head on the pillow. The second was that of a man who if he lay with his head turned to the right showed rotatory nustagmus on deviating the eyes to the right and vertical nystagmus on deviating them to the left. Bárány showed that the occurrence of the nystagmus depended on the posture of the head, and was not affected by the posture of the neck (relation of head to body).

Since then numerous similar observations have been recorded. Buys and Hennebert at a discussion in the *Groupement Belge otoneuro-ophtalmologique* emphasise that there are many sources of error to be guarded against, of which the chief is that nystagmus may be caused by displacement of the head relative to the trunk, or by the movement involved in bringing the head into the new position for examination. A case in which nystagmus resulted from changing the position of the head relative to the trunk has been recorded by de Kleyn and Versteegh. In some other recorded cases nystagmus manifested itself both after changing the position of the head in space and after changing it in relation to the trunk. It has been shown that in animals rotation of the head on the trunk may interrupt the circulation in one vertebral artery and thus in occasional cases it may induce circulatory disturbances in the labyrinth on the same side and consequently nystagmus. Variations of the positions of the head relative to the body must therefore be avoided in examining for real postural nystagmus. Secondly the movement of changing the position of the head must be performed very slowly.

The nystagmus seen in these peculiar cases is usually rotatory on deviation of the eyes to the lower side and vertical on deviation towards the upper; it usually appears after a short latent period and persists only a few seconds or minutes; its amplitude is small and its occurrence with successive trials is in many cases inconstant. There has been a tendency to connect the origin of this postural induction of nystagmus with the labyrinthine postural reflexes on the eyes described by Magnus and de Kleyn. This view was combatted by Professor Quix, who pointed out that these latter reflexes were tonic or lasting reactions. Quix attributes the postural induction of nystagmus in certain cases to a functional disturbance of the semicircular canals which is accentuated in certain positions by the normal action of the otoliths on the maculæ of the saccules. Quix has described an apparatus in which patients can be examined with the head in any angular position in space; it consists of a large circular frame mounted at each end of its horizontal axis on a stout tripod, and capable of being rotated round this axis and fixed in any position;

within this frame on a diameter at right angles to the axis of rotation, a much narrower oval frame, like the top of an operating table, is fixed, and this is capable of rotation round its long axis and can be fixed in any position. The patient is fastened on this smaller frame in such a way that he feels perfectly secure. The pressures of the otoliths have been calculated and their pressure for the head in any given angular position can be expressed as a percentage of their maximum pressure. J. P. M.

[27] **Amnesia, epileptic convulsive seizures and hemiparesis as manifestations of insulin shock.**—W. Lindsay Miller and J. H. Trescher. *Amer. Jour. Med. Sci.*, 1927, clxxiv, 453.

To the commoner symptoms of insulin shock the authors add certain hitherto unrecorded signs of nervous system involvement. Their patient had been having 90-100 units of insulin a day before entering hospital.

While in hospital he manifested, in addition to the usual symptoms of insulin shock, speech disturbance, some degree of amnesia, and generalised tonic-clonic convulsions in one of which he bit his tongue. In mild attacks his vision was blurred; in a severe attack the pupils became dilated and inactive to light. After one severe attack with convulsion, he showed a right hemiparesis and a diminution of the abdominal reflexes. They adduce evidence which goes to show that hypoglycæmia is not the only factor producing shock symptoms, for these may come on in the absence of hypoglycæmia and be relieved without glucose therapy.

They also suggest that shock symptoms are more prone to occur in persons with unstable sympathetic systems, and that the reduction in the inorganic phosphates and potassium of the blood caused by insulin may be an additional factor in producing such symptoms. They draw a tentative parallel between the disturbance of intermediary metabolism known to occur in tetany, and this condition.

P. W.

[28] **Subacute diffuse ependymitis.**—J. H. Globus and I. Strauss. *Arch. of Neurol. and Psychiat.*, 1928, xix, 623.

In the case reported by the authors the outstanding clinical features were the manifestations of increased intracranial tension, including almost all of the cardinal signs and symptoms of cerebral neoplasm, such as attacks of giddiness, intense headache, projectile vomiting and papilloedema. These signs and symptoms, with the gradual unfolding of the clinical picture, interrupted by slight remissions, and the absence of febrile reaction, justified a diagnosis of tumor cerebri. In the absence of well-defined focal signs the bilaterality of symptoms suggested a median line location, possibly between the hemispheres. Ventriculographic studies and the injection of dye revealed the presence of bilateral, symmetrical dilatation of the lateral ventricles and demonstrated conclusively the lack of intercommunication between the third and lateral

ventricles. These observations definitely localised the lesion and placed it in the third ventricle, but the true nature of the disease was not recognised during the life of the patient.

Microscopic investigation indicated the presence of an inflammatory process limited mainly to the ependymal lining of the entire ventricular system of the brain. The mild inflammatory and degenerative changes seen in the adjacent brain tissue were little more than a reaction to a contiguous inflammatory process. In the widespread ependymitis there were present all gradations, from the mildly acute purulent in the lateral ventricles to the chronic granular ependymitis in the aqueduct and the fourth ventricle. The third ventricle was occupied by a pseudocyst formed of thickened ependyma.

The presence of several transitional stages in the inflammatory process in one and the same case supports the belief that many of the so-called chronic forms of ependymitis, including granular ependymitis, especially when they are associated with an inflammatory process elsewhere in the central nervous system, as in general paralysis, are the results of a slowly progressive form of inflammation of the lining of the ventricular system.

R. M. S.

[29] **Aphasia ; a study of normal control cases.**—G. H. J. Pearson, B. J. Alpers and T. H. Weisenburg. *Arch. of Neurol. and Psychiat.*, 1928, xix, 281.

The authors have studied the nature of the responses that normal persons give when the tests used in the study of aphasia are applied to them. The control material consisted of two main groups : persons with superior and average intelligence without organic nervous disorder, and persons with organic brain disease of different types. The subjects in both these groups were without speech disorders. In selecting the control material, an attempt was made to obtain young persons without nervous disease, in order to perceive what reactions healthy, non-aphasic persons would give. In addition, however, patients with organic brain disease but without disturbances of speech were selected in order to approach more closely the condition of the aphasic patient who has brain disease of a highly localised nature. The tests employed were those devised by Henry Head.

It was found that the normal person often gives responses which are exactly the same as those obtained in cases of aphasia. In some tests the errors were so frequent and coincided so closely with those in aphasic cases that it was deemed wise to discard these tests. Although it is impossible to separate in any way the function of speech from intelligence, the results with Head's tests indicate that they investigate intelligence to a far greater degree than they do speech, and the conclusions drawn from them must stress, automatically, the loss in the former sphere rather than in the latter. The authors conclude that any other series of tests would have given identical results,

but they consider the tests used by Head to be as good as, if not better than, any others that have been devised for the testing of aphasia.

R. M. S.

[30] **Pellagra: a clinical study with notes on some cases.**—S. J. CANTOR. *Med. Jour. Austral.*, 1927, xiv, 713.

THE author records five cases of pellagra. He discusses briefly the various theories as to the mode of origin of the disease, and adopts the vitamin deficiency hypothesis.

He points out that the diagnosis cannot certainly be made without the triad of alimentary, cutaneous and nervous symptoms. The rash is the most important diagnostic sign, and he stresses its symmetry, its sharp delimitation, the branny desquamation and its predilection for certain sites. The subsequent pigmentation tends to be chronic and patchy.

That the cerebrospinal fluid is normal in spite of the gross cellular changes in the cord is against an infective hypothesis. Amnesia, mental retardation and tremor are among the early mental symptoms; the later are usually melancholia or confusional insanity with suicidal tendencies.

In most of his cases he was able to find evidence of faulty diet, and better dietaries, including eggs, fresh meat and milk and green vegetables caused immediate improvement. The common occurrence of the disease in institutions and among the poorer classes is correlated with dietetic faults in both cases.

P. W.

[31] **Sympathetic irritation in a case of actinomycosis.**—R. M. BRICKNER and A. P. ZEMANSKY. *Arch. of Neurol. and Psychiat.*, 1928, xix, 515.

A CASE of actinomycosis of the right lung and right posterior chest wall is described, in which disturbances of function of the thoracic sympathetic chain occurred; they were probably irritative. The area in which sympathetic changes were manifested included the right side of the face and neck, the entire right upper extremity and the right side of the chest down to the twelfth rib. The midline and the line of the twelfth rib formed sharp boundaries.

The sympathetic changes were indicated by localised hyperidrosis, appearance of goose-flesh, pilo-erection and unilateral nipple erection, an exaggerated response to the drawing of a pin across the right side of the front of the chest, a lowering of the temperature, with pallor of the right hand as compared with the left, and bizarre responses of the right upper extremity to extensive changes in temperature. In a general way, the reactions of the right arm and hand were the opposite of those of the left.

No difference between the right and the left upper extremities were observed in blood pressures, growth or condition of the hair or nails, reactivity of muscles or nerves to electrical stimulation, nor in their reactions to epinephrine.

Permission for an autopsy could not be obtained. R. M. S.

[32] **Rabies in man.**—K. Lowenberg. *Arch. of Neurol. and Psychiat.*, 1928, xix, 638.

Working at the psychiatric clinic of the university of Minsk, Lowenberg had the opportunity of studying the microscopic appearances in four cases of rabies, and in general was able to confirm the observation of Schukri and Spatz that the inflammatory changes in rabies are of a type and localisation quite similar to those found in cases of lethargic encephalitis.

The brunt of the attack seems to be borne by the substantia nigra and the substantia centralis grisea and, to a lesser extent, by the pontine nuclei. In all of these the reactive phenomena are strikingly localised about the parenchyma, the nerve cells being largely destroyed and accompanied by neuroglial proliferation and neuronophagia. In the cortex, basal ganglia, white matter and cerebellum the lesions are of a predominatingly degenerative character.

This picture was especially characteristic of the author's first two cases, whereas cases 3 and 4 showed some deviations. In case 3 the picture was that of a meningitis, there being leucocytic infiltration, marked œdema and occasional hæmorrhages. In case 4 the appearances were those of an acute purulent meningitis, but without abscess formation. These four cases demonstrate that there is a relation between the length of the period of incubation and the character of the lesion, for the shorter this period, the more acute and intense is the process. Thus, in the first two cases, with incubation periods of over six months, there was no meningitic involvement and the degenerative process was more distinct. In case 3, with an incubation period of a little more than a month, the inflammatory reaction was more pronounced, while in case 4, in which the incubation period was only nineteen days, an intense inflammatory process was present.

A further point of interest, which appears to have escaped the author, is the relation of the site of the infected wound to the incubation period. In cases 1 and 2 the bites or scratches were inflicted on the hands, in case 3 on the hands and chest, and in case 4 on the face. The short incubation period in cases 3 and 4 is exactly what one would expect in a disease having all the characters of a lymphogenous infection of the nervous system.

R. M. S.

[33] **The 'cog-wheel' phenomenon in Graves', physiopathic, and vestibular syndromes** (Le phenomène de la roue dentée dans les syndromes Basedowiens, physiopathique et vestibulaires).—F. Negro. *Revue neurol.*, 1928, i, 502.

Dr. F. Negro claims to be able to demonstrate the occurrence of the 'cog-wheel' phenomenon in various syndromes distinct from those of Parkinsonism, in which it was described originally by C. Negro in 1902. In these its degree is less, yet it is distinctly recognisable. His argument is to the effect that the phenomenon is the outcome of sarcoplasmic rigidity or hypertonicity; assuming

that the sarcoplasm is innervated by the parasympathetic, he states that the supposition would be that sarcoplasmic rigidity follows from the stimulating action of thyroid secretion on the parasympathetic; were this the case, however, it should be more pronounced in vagotonic than in sympathicotonic states. But the reverse is the case. The conclusion that sarcoplasmic innervation must be from the sympathetic and not the parasympathetic seems the natural one to make. The author, however, brings forward evidence suggesting that the thyroid secretion has a double effect, on the two divisions of the system.

J. V.

[34] **Haemangioma of the spinal cord.**—C. W. RAND. *Arch. of Neurol. and Psychiat.*, 1927, xviii, 755.

THE author reviews the literature of twenty-one cases of hæmangioma of the spinal cord, and describes two new cases.

The majority of hæmangiomas have been found on the dorsal aspect of the cord and it is supposed that they originate from the pial veins. They may be seen in the cervical region, although the lower dorsal and lumbar regions are the locations of choice. The vessels may enlarge to enormous proportions and may present a nest of greatly dilated, closely packed, interwoven veins which compress the underlying cord at times almost to a point of flatness. On occasion, the varicosities may extend into the cord itself, and the nutrition of this structure becomes so impaired that areas of softening result.

Spontaneous rupture is prone to occur and is cited as the most common cause of sudden paraplegia. The rupture may be free into the spinal canal, or it may be intramedullary. Thrombosis has been reported in a number of instances and is perhaps more common that spontaneous hæmorrhage. It may affect the vessels within or without the cord proper. In certain instances the blood supply to the surrounding vertebræ may be greatly increased, and the veins of the adjacent soft tissues dilated almost to angiomatous proportions.

A clue to the diagnosis is sometimes afforded by the presence of a nævus or multiple nævi in the dermatome supplied by the cord segment from which the pial hæmangioma arises, and when present it is deeply pigmented.

Surgical intervention offers little hope of improvement in these cases.

R. M. S.

[35] **Malignant chordoma of the lumbar region.**—C. DAVISON and A. WEIL. *Arch. of Neurol. and Psychiat.*, 1928, xix, 415.

A CASE of malignant chordoma at the third lumbar vertebra is reported. This is the second case of its kind out of fifty-eight published case reports.

Tumours of the notochord are difficult to differentiate from myxoid chondroma and colloid carcinoma of the intestinal canal. Usually chordomas are soft and gelatinous in consistency, and clinically are often mistaken for abscesses, but the tumour described in this paper was of fibrous consistency

and of a mixed character. In its outer layer it resembled a sarcoma, but the physaliphorous cells and the staining quality of the matrix characterised the tumour as a chordoma.

R. M. S.

[36] **Retractile myosclerosis of old age and its association with akinetic-hypertonic syndromes (pallidal syndromes—Foerster's arteriosclerotic rigidity)** (La myosclérose rétractile des vieillards et ses rapports avec les syndromes acinéto-hypertoniques).—J. LHERMITTE. *L'Encéphale*, 1928, xxiii, 89.

No one has done more than Lhermitte to throw light upon the various types of senile paraplegia. Over twenty years ago, in collaboration with Lejonne, he isolated a clinicopathological group in which muscle changes alone occupy the picture. Under the title of " myosclérose rétractile " (or " myopathie primitive sénile ") Lhermitte refers to a type of slowly progressive paraplegia without any corresponding affection of the central or peripheral nervous systems. Despite its frequency in institutions for the aged, but little attention has been paid to this condition. The onset is usually seen in extreme old age ; women are affected more often than men. Pains, cramps and other dysaesthesiæ localised to the calves usually constitute the first symptom. Later appears difficulty in walking and some months afterwards the patient became bedridden. Thereafter the course is more rapid ; muscles waste and shorten, and the lower limbs assume an attitude of flexion. At first the legs can be passively extended, but in time this becomes impossible even under anæsthesia. The first picture is that of extreme paraplegia in flexion ; the muscles are profoundly wasted and feel hard on palpation ; the tendons stand out like violin strings. Both active and passive movements are impossible, and attempts at manipulation are very painful. There is no marked change in the electrical reactions, but mechanical excitability is increased and myoidema is constant. The upper extremities and the neck and trunk usually escape. Cremasteric and abdominal reflexes are absent ; the knee jerks are often sluggish and it may be difficult to elicit an ankle jerk. There is never a Babinski response. Lhermitte discusses the differential diagnosis from arteriosclerotic rigidity, with which indeed it is frequently associated., Jakob's disease, or senile *Vorsteifung*, is regarded by Lhermitte as merely a variety of arteriosclerotic rigidity. Histological changes in senile myosclerosis are profound ; they comprise atrophy, pigmentation and plasmodial regression of the muscle-fibres with fatty, pigmentary and other types of degeneration ; colossal increase of the inter- and intra-fascicular connective tissue, with replacement of muscle fibres by fatty or fibrous elements ; some of the fibres are hypertrophied. The lymphatic spaces are distended and there are accumulations of small round cells. The genesis of the muscle changes is thought to depend not solely upon an inadequacy of the peripheral circulation but to embrace also a stagnation in the lymphatic

flow from the muscles, together with a disorder of general nutrition. These factors are exaggerated by the forced immobilization of the limbs.

The paper is finely illustrated. M. C.

[37] **Vasomotor and pilomotor manifestations.**—Temple Fay. *Arch. of Neurol. and Psychiat.*, 1928, xix, 31.

The author calls attention to the localising value of vasomotor manifestations in lesions of the spinal cord.

The patient is placed in diffuse daylight of moderate intensity, the possibility of shadows being avoided, the covering is quickly removed, and the entire body scanned carefully. This is best done at a distance of from three to six feet from the patient, and careful attention should be paid to the presence of areas of hyperæmia, which are suggested by a broad band of flushing about the chest and abdomen, demarcated above by the normal flush of the body. The direction of this band of vasomotor manifestation is in striking accord with the segmental zones of Head. Several variations of the phenomena may exist, and all have been found to be of equal localising significance, indicating an abnormal instability in this area. The skin area above and below the line of demarcation may vary in texture and in the degree of light reflection, if the patient is observed from an angle such as from the head or from the foot of the bed. The actual line of demarcation may assume a feather-edged appearance and may be no larger than the diameter of a piece of twine, extending in a serpentine fashion at right angles to the axis of the body until the axillary line is reached, and then coursing upward at an angle of about 30 degrees toward the spinous processes. The zone of demarcation in cases in which the history indicates a long-standing involvement of the cord may simulate the difference between a slight sunburn and normal skin, so that the area below the level of the lesion appears to be slightly pigmented, and this effect may extend over a large area of skin below the line of demarcation, giving the appearance that the patient had been over-exposed to the weather.

The examiner will be struck by the rapid evanescence of the vasomotor reaction in the zone under observation. During the initial period of observation a flushing should appear at the level noted, similar to that seen in slight hyperæmia following the application of heat to the skin. If the patient is exposed to a draught or cold, it may be noted that this area blanches rapidly, giving rise to an apparent ischæmia or to a zone distinctly white in comparison with the normal areas above. Confirmation of the level of vasomotor demarcation can be intensified by heat, and the zone will be found to coincide exactly with the first signs of sensory change. Pilomotor manifestations can be obtained by flicking the skin with a wet towel or by the application of a current of cold air. Similar results can be obtained by deep pinching of the skin and the trapezius muscle at the base of the neck; this produces a homolateral reflex

spreading rapidly down the same side to include the lower extremities. This is a test for function of the cord and may give the level of the lesion by failing to appear below the involved segment.

In the author's series of thirteen cases the vasomotor line of demarcation proved to be the most reliable sign, and in each case it determined the exact level of localisation. R. M. S.

[38] **Clinical studies on the question of the value of the law of Bell-Magendie** (Klinische Studien zur Frage der Geltung des Bell-Magendieschen Gesetzes).—R. WARTENBERG. *Zeits. f. d. g. Neurol. u. Psychiat.*, 1928, cxiii, 518.

DR. WARTENBERG's long and valuable discussion of problems connected with the function of dorsal and ventral spinal roots does not lend itself to a convenient summary. It is replete with analyses of numerous personal and recorded cases, and covers various separate if cognate questions each one of which is more or less controversial. His investigations have extended over a number of years and concern diverse clinical material. The paper is full of useful references to the literature.

The author is at pains to impress the reader with the uncertainty attending many of the " conclusions " that have hitherto been utilised as data on which to base current conceptions of the function of anterior and posterior roots. Some of his own decisions may be briefly indicated.

After section of posterior roots (several consecutive roots on one side) all forms of cutaneous sensibility are lost, but a degree of deep sensibility remains. To determine its presence the examiner must use stimuli of sufficient strength and duration. It appears definite enough that this deep sensibility is not as a fact identical with that which can be demonstrated on the normal side ; sensations of pressure and of pressure-pain arise more slowly than normal, and tend to remain for an appreciable moment after cessation of the stimulus ; they have a high threshold ; as a rule, the sensation is less painful than normal, more diffuse, more blunt—in a word, it is but a fraction, so to speak, of normal deep sensibility. Yet stimuli have local signature. It undoubtedly presents in these respects some resemblance to so-called visceral sensibility ; a summation of stimuli is requisite, in regard to both number of end-organs stimulated and duration of stimulus. The author is evidently impressed with this resemblance, which embraces both clinical characteristics and conjectural site of origin.

A further conclusion is to the effect that such excitations must reach the cord by anterior roots. Other possible routes are examined at length, but rejected for want of evidence. The author exercises admirable scientific restraint when he says that the path *may* be by anterior roots, not *must*. In different parts of the same nervous system, and for different individuals, the amount of sensibility thus conveyed by anterior spinal roots or cranial nerve analogues varies materially. S. A. K. W.

[39] **Icterus neonatorum gravis. Report of a case which recovered with sequelæ pointing to an involvement of the central nervous system.**—H. M. GREENWALD and W. MESSER. *Amer. Jour. Med. Sci.*, 1927, clxxiv, 793.

THE authors record the case of an infant which recovered from icterus neonatorum gravis. The infant was the third child of the family. The two elder children had died of the same disease. The delivery of the third infant was normal in every way, but it was deeply jaundiced within twenty-four hours of birth. This jaundice persisted up to five weeks of age, during which time, although the stools and urine contained bile pigment, the child's weight curve was normal for its age. There was no fever. The Wassermann reaction of the mother and child was negative. Bleeding and coagulation times were normal.

At six months the child could not support its head and showed spasticity of the thigh adductors. At two years old the child was idiotic in appearance, could only utter a few monosyllables, and could only walk on its toes with support. The gait was spastic and the plantars double extensor.

The authors review the suggestions as to the pathogenesis of this condition, and feel that sepsis and maternal toxæmia are not likely to have played any part in their case. They quote the results of autopsy which have been recorded up to the present. Necrosis of the ganglion cells of the basal nuclei and round-cell perivascular infiltration were found in two cases by Hoffmann. Icteric pigmentation and necrosis of the ganglion cells of the basal nuclei, the medulla, the olivary bodies and the gray matter of the anterior and posterior horns of the cord have been found in newborn infants dying of the disease. Pigmentation is not, however, constant. The liver, in some cases, has shown diffuse necrosis and destruction of cells most marked in the centre of the lobules. Cerebral hæmorrhage or injury was found in no case. The two outstanding features in symptomatology are motor disturbances of different kinds and intellectual defect.

The authors follow Hoffmann and Hausmann in believing that cerebral changes only occur where there has been definite necrosis of liver cells. Cholæmia as such cannot produce these changes for they are not found in congenital atresia of the bile ducts, or in all cases of icterus gravis. They support this view by adducing the cerebral symptoms which occur in acute yellow atrophy, and the brain changes found in infants suffering from septic icterus and septic nephalitis. In both these conditions necrosis of liver cells does occur.

They therefore consider, that icterus neonatorum gravis must be regarded as a possible cause of some cases of cerebral diplegia. P. W.

[40] **Myatonia congenita of Oppenheim.**—J. M. NIELSEN. *Amer. Jour. Dis. Child.*, 1928, xxxv, 82.

THE author recognises two forms of myatonia congenita—the commoner occurring in one member of a family and causing early death ; the other being

one-tenth as common, familial, and benign in that the patients survive to puberty and may even recover.

He records a case of the latter type, that of a boy, age seven. The parents were healthy but the mother had had one anencephalic infant, and one still-birth at three months. The patient was the third child, the two next children were healthy, and the sixth pregnancy resulted in a miscarriage at eight months.

The patient was never able to hold his head up properly and had always walked with a waddle from the age of 14 months. He talked normally and was mentally up to the average. The head was held constantly retracted owing to absence of the sternocleidomastoids and overaction of the posterior nuchal musculature, which was in a state of contracture. Cephalic rotation was almost impossible ; the child moved his eyes only to look to the sides, and he had to use his hands to bend his head forward. This contracture readily accounts for his waddling gait, and the constant gape. No microscopic examination was made of the neck muscles.

The other features on which the diagnosis was based were the generalised small musculature, the muscular flaccidity, the absence of all deep reflexes except on reinforcement and the abnormal mobility of all joints. The author brings the neck contracture into line with the talipes equinus and the lamb contractures so often seen in amyotonia congenita, but he considers it to be thus far unique.

P. W.

[41] **Toxic infectious disease in the peripheral and central nervous system—so-called neuronitis.**—S. Brock and M. Ivimey. *Arch. of Neurol. and Psychiat.*, 1928, xix, 129.

A series of five non-fatal cases is presented, illustrating toxic or infective processes involving the peripheral nerves and the central neuraxis. This motley group, with a varied etiology, showed a mixed symptomatology referable to the peripheral and central nervous system.

The authors suggest a nomenclature in which the term neuritis is retained—peripheral neuritis for processes limited to the distal parts of the nerve trunks ; radiculitis, for involvement of the roots, and central neuritis (spinal, medullary, pontine, etc.) for involvement of the neuraxis. When more than one part of the nervous system is implicated, these terms may easily be compounded.

R. M. S.

[42] **A case of essential thrombopenia (morb. Werlhof) with haematomyelia.**—Karl Evang. *Acta Psychiat. et Neurol.*, 1928, iii, 7.

The case reported appears to be the first clinical instance recorded of spinal hæmorrhage in thrombopenic purpura.

The patient was a man of 30, with a pronounced hæmorrhagic diathesis (full details given), in whom a flaccid paralysis of both lower extremities and of the muscles of the abdomen, as well as loss of sensibility up to the second

rib, set in suddenly without ascertainable cause. During six months of observation the condition passed from the stage of flaccid paraplegia, through spasticity with flexion contractures, to spasticity in extension.

The pathological diagnosis is discussed at some length and finally regarded to be one of hæmatomyelia. The findings in the spinal fluid were curiously indefinite. As for the blood picture, its features were: small number of platelets (less than 35,000); non-retraction of the coagulum; prolonged periods of bleeding; almost normal coagulation-time; positive stasis test; extravasations of blood on contusion—all typical of essential or benign thrombopenia.

J. V.

[43] **Chvostek's sign in the newborn.**—Stevenson, Mitchell and Koch. *Amer. Jour. Dis. Child.*, 1927, xxxiv, 425.

The authors determined the incidence of Chvostek's sign in a series of 55 infants ranging in age from 12 hours to 21 days. All the infants had a positive Chvostek's sign. They considered the sign positive when a contraction was obtained at the lip, alæ nasi or forehead.

Blood calcium estimations were normal in all cases, and other signs of tetany such as Schultze's, Lust's and Trousseau's were not elicited.

The authors stress the fact that the sign cannot be elicited in a restless or crying infant.

They conclude that as Chvostek's sign can be demonstrated in newborn infants with no evidence of tetany, it is not to be looked on as pathognomonic of this condition in early infancy.

P. W.

[44] **Diaphragmatic paralysis resulting from injury of the brachial plexus.**—J. W. Epstein. *Amer. Jour. Dis. Child.*, 1927, xxxiv, 634.

The author records the case of a female infant, age three-and-a-half months, showing unilateral phrenic palsy. The labour had been difficult, a breech presentation in a primipara, and the infant sustained a fracture of the left humerus and a right-sided Erb paralysis.

On examination he found the respirations rapid and irregular, and the middle of the sternum retracting with each inspiration. The right half of the chest was flattened and moved less well than the left. There was dullness at the right base. The abdominal respiratory movements were completely absent on the right side, and an X-ray examination showed a paradoxical ascent of the right half of the diaphragm in inspiration.

The child was watched for two and a half years during which time it had three attacks of right-sided bronchopneumonia.

The author reviews the literature on phrenic paralysis. Few cases due to injury are on record owing to the well protected position of the nerve in the neck.

Obstetric injury to the phrenic nerve was first recorded in 1921 by Kofferath. It has never been seen without concurrent injury of the brachial plexus, and recovery has occurred in the other two recorded cases.

The symptoms are all referable to unilateral diaphragmatic paralysis, but are more severe in the newborn as a state of atelectasis exists on the affected side.

P. W.

[45] **A case of hemihypertrophy** (Un cas d'hemi-hypertrophie).—VERMEYLEN. *Jour. de Neur. et de Psych.*, 1928, xxviii, 103.

THE recorded cases of hemihypertrophy number 53. To these Vermeylen adds the case of a young girl of 12 in whom the left side was considerably larger than the right. This hemihypertrophy involved face, ear, tongue, neck, breast and limbs. The hemicircumference of the trunk on the left side was six centimetres more than on the right, the circumference of the left calf and that of the left thigh were each four centimetres greater than the corresponding measurements on the right. The left leg was six centimetres longer than the right. The hands and feet and genital organs showed no asymmetry. The girl was mentally defective, her intellectual development being that of a child of seven.

The most probable explanations of hemihypertrophy are (1) that it results from a tendency to twinning in the ovum (Gesell), and (2) that it is a consequence of an abnormality in the development of the sympathetic system on one side (Leustrup).

J. P. M.

[46] **Liver dysfunction in migraine.**—J. S. DIAMOND. *Amer. Jour. Med. Sci.*, 1927, clxxiv, 695.

THE author describes his investigations in a series of 35 cases of migraine. Cases which showed other pathological changes were excluded.

A very complete examination was made, and in 32 out of his 35 cases he demonstrated a bilirubin retention varying from 1 to 5 units as estimated by the van der Bergh reaction. The urobilinogen in the urine varied, but was found constantly raised in those cases with a high bilirubin retention.

Most of his cases gave a familial history and in most the headaches had begun in the first or second decade. All showed headache of the hemicrania type.

The van der Bergh reactions were lowest in the purely cephalic type, higherl in those showing intestinal disturbance, and highest in the cases of abdominal migraine. In the latter cases, the symptomatology is identical with that of cephalic migraine in the early part of the attack ; intense epigastric pain and vomiting of bile characterise them in their later stages.

All his cases showed a low systolic blood pressure, 90-110, and constipation.

He considers that a failure on the part of the liver to synthesise and detoxicate the amino bodies formed during digestion leads to a series of anaphylactic changes in the cerebrum and other viscera analogous to those occurring in Quincke's œdema. Treatment should be directed to overcoming the constipation, and protecting the liver by avoiding animal protein and giving a lacto-vegetarian diet.

P. W.

PROGNOSIS AND TREATMENT.

[47] **Arsenical neuritis treated by the intravenous injection of sodium thiosulphate.**—T. Dixon Hughes. *Med. Jour. Austral.*, 1927, xiv, 543.

The author reports a case where signs of chronic arsenical poisoning followed on taking a dose not far short of the fatal one. Intense purging and collapse followed on the ingestion of the dose. Five days later arsenical dermatitis appeared, twelve days later paræsthesiæ in the hands and feet and seventeen days later weakness in the arms and legs. Seven months later there was still definite objective sensory loss in the hands and feet, tenderness of the calves on pressure, the knee jerks were absent and there was wasting of the small muscles of the hands and of both thighs.

Arsenic was found in the urine. The patient was given 16 injections intravenously of 0·75 gr. of sodium thiosulphate on alternate days. Arsenic had disappeared from the urine within three weeks, and at the conclusion of treatment he was practically normal.

The rationale of the treatment is that the sodium thiosulphate forms a soluble arsenate which is rapidly excreted, thereby displacing the relatively insoluble arsenious acid compounds.

P. W.

[48] **Atypical neuralgia.**—C. H. Frazier. *Arch. of Neurol. and Psychiat.*, 1928, xix, 650.

Removal of the superior cervical ganglion and periarterial sympathectomy do not appear to be of much value in the treatment of atypical neuralgia, mostly in the trigeminal distribution. The author presents the data from ten selected cases, in five of which the sensory root operation had first been performed. In only one case did the patient obtain any relief, and a doubt remained as to whether the relief was permanent, as the patient could not subsequently be traced.

R. M. S.

Endocrinology.

[49] **Growth of the thymus.**—E. Boyd. *Amer. Jour. Dis. Child.*, 1927, xxxiii, 867.

The author reviews the earlier conceptions as to the growth and pathology of the thymus. Her investigations were made on a series of 147 children, ages from two weeks to 14 years.

She finds that Paltauf's original picture of status thymicolymphaticus corresponds with the normal development of the thymus and lymphoid tissue in well-nourished children. Her investigations support Hammar's conclusions, that the thymus normally increases in weight up to puberty and then undergoes an age involution. She has also shown that the thymus weight fluctuates with the body weight, diminishing in acute infections, and being greater in well nourished than in poorly nourished children. There is a definite parallelism between the thymus growth curve and that of the lymphoid tissue generally. Confusion has arisen from failure to recognise that accidental involution of the gland may occur in infections.

In discussing mors thymica she states that in none of her cases was death caused by pressure of the thymus on the trachea. She finds that a normal gland may produce symptoms by pressure on adjacent structures, especially the recurrent laryngeal nerve. That these symptoms do not appear until the fourth week of life is explained by the fact that the thymus joins in the normal loss of body weight seen in the first ten days of life ; that they do not appear after the first year of life by the fact that the chest is growing more rapidly than the gland which, therefore, becomes mechanically insignificant. P. W.

[50] **The mongol : a new explanation.**—R. M. Clark. *Jour. of Ment. Sci.*, 1928, lxxiv, 265.

Disease of the thyroid gland is known to cause : (*a*) Myxœdema ; (*b*) its congenital form cretinism, associated with hypothyroidism ; and in contrast (*c*) exophthalmic goitre, associated with hyperthyroidism. In the view of the writer it is possible to add a fourth type, the mongol, which he believes to be a congenital form of exophthalmic goitre. Few similarities between this condition and mongolism are obvious. Hyperthyroidism in the adult causes definite symptoms, and were such a condition to exist in a rapidly developing fœtus and cease at birth—even as a sudden and complete remission may occur in exophthalmic goitre—the resulting infant would, it is believed, at birth present the features of mongolism. In support of such a theory, one must bear in mind the responsiveness to environment and altered conditions during growth, of all structures, including muscle, tendons, glands, skin, etc. It is conceivable that antenatal hyperthyroidism causes the general muscular hypotonus and explains the pot belly, hernia, over-flexibility of the limbs and low blood-pressure of the mongol. On a closer examination of the problem it seems remarkable and more than a coincidence that in both conditions so many similar

points are attacked, even although the results of the attack differ. A more careful examination of the condition of the eyes—a characteristic feature in both diseases—reveals the reason for the different results. From intrauterine protrusion of the eyeballs there would result stretching of the coverings of the eyeballs and of the developing ocular muscles and pressure on the eyeballs. This would account for such features as the thin eyelids, the high arched brows, small eyes and liability to polar cataracts, and the defective and falling-out eyelashes. Stretching of the tissues over the bridge of the nose would explain the absence or ill-development of the nasal bones with flattening of the bridge of the nose, and the stretch and pull from this region would account for the short squat nose, while the retraction of the point of the nose would explain why the nostrils look forward and are narrowed. The increased intraorbital pressure would explain the thinning of the orbital bones and, if the protrusion of the eyes ceased at birth, the resulting slackness would ill fit the eyeballs, germs and dirt would be harboured, and cause the blepharitic and other inflammatory troubles so common in mongols. The slanting eye may be explained by antenatal hyperthyroidism causing eyeball protrusion and contraction of Landstrom's muscle in the fœtus. The undersized and stunted growth is the result of changes in the eyes, the hands, the feet, the vertebral column, skull and brain. In the mongol ontogeny has stopped short at a late stage in the life history of the human fœtus, and yet development has gone on to maturity and viability. Thyroid experiments with tadpoles seem to confirm this theory. Other conditions as well as mongolism have been attributed to endocrine disturbances by some authorities, by others to "reversion." It is more likely that a theory which incorporates both views is the correct one.

C. S. R.

[51] **An investigation of the effect of glandular therapy on the intelligence quotient.**—Edna J. Fox. *Mental Hygiene*, 1928, xii, 90.

A positive correlation was found to exist between dysfunction and I.Q. when a glandular-dysfunctioning group, whose average I.Q. was 74, was compared with an unselected clinical group whose average I.Q. was 78.

When the glandular dysfunctioning groups were separated, the lowest correlation between dysfunction and I.Q. seemed to exist in the hyperthyroid group and the highest in the hyperpituitary group. The other groups presented a correlation similar to that of the unselected clinical group. It should be noted that many of the groups were too small to be really significant.

From a re-examination for intelligence-quotient ratings, it would seem that the pluriglandular cases made the greatest appreciable gain in I.Q. after glandular therapy was administered. This gain was 4 points. It did not appear that there was any appreciable gain in I.Q. rating after glandular therapy, especially in the hyperpituitary cases, in which an actual loss of two points occurred. The average net gain in I.Q. points when all cases were considered was 1·5 points. The average gain in points when the loss was not taken into account was 3.5 points per individual.

C. S. R.

[52] **Neuropsychiatric aspects of lipodystrophic disturbances.**—Lloyd H. Ziegler and Curtis T. Prout. *Amer. Jour. Psychiat.*, 1928, vii, 709.

Eighty-two reports of cases of lipodystrophy are here reviewed. Fifteen patients were markedly self-conscious about changes in their appearance. In 17 cases the families or friends were worried about the patient's health. In 8, nervous or mental symptoms were rather marked. In 43 no abnormality of behaviour was noted. There seemed to be no psychiatric symptom-complex common to lipodystrophy, aside from self-consciousness and the tendency to share in the alarm of friends and relatives. Psychiatric symptoms when present seemed on the whole to be psychoneurotic. No constant neurological symptoms or signs were observed accompanying lipodystrophy. Although weakness is a usual symptom at one stage or another of lipodystrophy, no general symptom-complex or diseases can be closely related to it until more cases are available for study. Nervous symptoms in all cases resulting from self-consciousness over personal appearance are not constantly present. No conclusive evidence has been collected to support the theory that the integrity of the nervous system is affected by the disorder.

C. S. R.

[53] **Parathyroid therapy in tetany.**—A. H. Gibson. *Amer. Jour. Dis. Child.*, 1927, xxxiv, 835.

Gibson records four cases of infantile tetany which were treated with parathyroid extract given hypodermically. In three of the cases the rise which occurred in the serum calcium and phosphorus was definitely related to the recovery from accompanying infection. The fourth case, which was complicated by thymus enlargement, proved rebellious to treatment of any sort.

He stresses the value of the accepted means for raising serum calcium and phosphorus—cod liver oil, calcium by mouth and quartz lamp irradiation—and he deprecates too great reliance on parathyroid extract alone.

P. W.

Psychopathology.

PSYCHOLOGY.

[54] **A theory of the smile.**—A. M. Hocart. *Psyche*, 1928, viii, 89.

The origin of the smile does not seem to have been as yet satisfactorily explained by psychologists. In order to trace the origin of anything we must have recourse to the comparative method, that is, we must collect all the different varieties and from them reconstruct a parent form that will explain them all. We have to search for the smile among other animals besides man. When a dog is pleased, especially when it is full of fun, it opens its mouth slightly, draws back the corners of the mouth, and bares its teeth. If it is tickled under the forelegs as it is lying, it is apt to draw back the corners of the mouth slightly

without baring the teeth. It thus reacts in much the same manner as man under the same conditions. An affectionate and playful puppy greets its master with ears cocked, wagging its tail, smiling, and rushes towards him and proceeds to chew his hand. Have we here the solution to the problem ? Is the smile merely a getting ready to bite and to do so without injuring ? The smile is the resultant of two opposite tendencies, the impulse to tear with the teeth and the impulse to suck and lick the object of affection. Adult man does not habitually bite the object of his affections, but there is a tendency which usually betrays itself only in words, and only in extreme excitement breaks out into action. In children there is a distinct tendency to chew. Kissing has, among Europeans, provided a platonic substitute. The parallel between the man's and the dog's smile is so close that the writer thinks we are amply justified in identifying the two. The dog's smile is more archaic ; in man it has been retained as a valuable index of the mind, and has therefore become more subtle and capable of expressing all kinds of shades which a dog's mind lacks. Secondly, the dog has not learnt to use the smile, or rather the emotion it indicates, as a protective reaction : the dog does not smile at anything unpleasant. If this theory is correct we should expect to find the smile among those animals which, when young, have to practice biting and tearing in play, not among others. As a matter of fact, herbiverous animals do not smile. The dog's behaviour may help us to explain the laugh as well as the smile. As he becomes excited his smile develops into a bark. When in earnest the bark is a signal to the enemy, " Keep away or I shall bite you." In play it threatens merely a mock attack. The laugh may thus be nothing but a bark tempered by affection ; it is to the angry bark what the smile is to the snarl. Man has given up barking in anger because he can express his threat more effectually in words ; but he has retained the playful bark as an expression of emotion which is not meant to convey any precise information. C. S. R.

[55] **The tooth as a folkloristic symbol.**—Leo Kanner. *Psychoanalytic Review*, 1928, xv, 37.

It is stated that the material brought forward demonstrates better than anything else what attention is paid and what importance is attached to the teeth all over the inhabited earth. No other part of the human body, with the exception perhaps of the eyes and the genital organs, disposes of such a wealth of folk-lore as do the teeth. The writer concludes that : (1) In folk-lore, the tooth is a sexual symbol, standing for both phallus and semen ; (2) the loss of a tooth, either accidental or physiological (dentition) or in the form of voluntary mutilation stands for castration, as punishment for onanism or other sexual perversities ; (3) toothache symbolises threatened castration—it is guarded against by measures which " make good " for the crime to be punished, by means of a symbolical performance of the normal sexual act with the aim of fecundation ; (4) the incest motive and bisexual fantasies recur frequently in the analysis of dental folklore. C. S. R.

[56] **Psychological effects of fasting.** — JOHN ARTHUR GLAZE. *Amer. Jour. Psychol.*, 1928, xl, 236.

As the results of experiments the writer concludes that :—

(1) So-called 'mental' out-put is generally decreased during a long fast.

(2) A fast of more than a week's duration definitely increases steadiness of hand.

(3) A subject fatigues more rapidly during a fast than normally, when the task is of considerable length and homogeneity.

(4) Performance at some tasks after a long fast is much more efficient than normally, an effect which can hardly be attributed to practice alone.

(5) In casual observations it was noticed (*a*) that smell sensitivity is greatly increased during a long fast ; and (*b*) that sex feelings in males are considerably accentuated when eating is resumed after a long fast.

C. S. R.

NEUROSES AND PSYCHONEUROSES.

[57] **The relationship between neuroses and disorders of the heart or blood-vessels** (Ueber die Beziehungen zwischen Neurosen und Herz- bezw. Gefässerkrankungen).—BERNHARD DATTNER. *Wien. klin. Woch.*, 1926, 1023.

THE patients coming to an out-patient department for "nerves" complain of many sensations for which our methods of examination show no organic cause. It is, however, probable, from the very uniformity of their complaints, that these may be evidence of dysfunction which is quite real, and fresh methods of investigation might enable us to understand the causes of the subjective symptoms, if we take them seriously and do not merely attribute them to excess of attention given by the patient to normal processes.

If we try to classify these complaints we find some prominent—palpitation, dyspnoea, giddiness, præcordial pain and vague anxiety—which point to circulatory disturbance ; these are brought on by exertion, alcoholism, emotional stress, etc., and suggest that the patient has a circulatory system which is not equal to standing stress. Physical signs found are rarely more than a slight irregularity of the pulse, occasionally tachycardia, and respiratory arrhythmia. Looking at the patient as a whole we find other associated signs, such as congestion of the face, damp and cyanosed extremities, transient erythema, cutis marmorata, and dermographia of varying degree up to urticaria factitia ; also arteries unduly hard for the patient's age, increased mechanical irritability of muscles, and in most cases occasional rises of temperature. These symptoms have been accounted for in a variety of ways, but not satisfactorily —they have been attributed to tubercular vascular effects, sometimes to the thyroid gland, and generally to a vague disorder of the autonomic system. Whatever be the real cause, there is evidently in these cases a circulatory

disorder affecting various localities, and probably also influencing tissue chemistry and respiratory exchange.

Neurotics have an inherited constitutional defect. It has been found that untreated cretins and other imbeciles show a high degree of failure in development of the normal form of the capillary network, while some degree of such failure is often present in other children ; parallel with this defect run others, such as persistence of infantile hair, late joining of epiphyses, and mental and physical maldevelopments generally. Where such defects of capillary development are found in adults, they are often associated with neurotic and arteriorenal disorders, and these are most frequent in regions of endemic goitre. Jaensch describes a parallel development defect of the capillary system and the organism in general. This view is supported by the benefit derived mentally as well as physically from the administration of thyroid and iodine in such cases.

It is probable that the defect in capillary development is due to the same ubiquitous toxic agency which makes goitre endemic, and it can be readily understood that people with an imperfect vascular system are more susceptible than others to stresses, whether mental or physical. The exhibition of thyroid and iodine is often beneficial, and as it relieves the anxiety as well as the bodily symptoms, it would seem that neurotic anxiety is largely based on physical sensations and may be a sort of external representation of a tissue-hunger for oxygen. It may be that the widespread use of iodine in many disorders is based on its beneficial effect on such circulatory disturbances in a variety of conditions.

If we look at neurotics thus, not as imaginary invalids having "nothing the matter," but as suffering from obscure disorders of metabolism and development which we should investigate, and try to put right, we shall no longer be content to administer a placebo and suggestion, but apply psychotherapy and physiotherapy alike with increased understanding and better results.

M. R. B.

PSYCHOSES.

[58] **Individual psychology and psychosis.**—Leonhard Seif. *Amer. Jour. Psychiat.*, 1928, vii, 639.

Adler's psychology which was originally developed in the treatment of the neuroses is here applied in order to arrive at an interpretation of psychosis and an understanding of the psychotic personality. Psychosis is a method of living, the mode of expression of a feverish ambition and unrealizable expectations on the part of a tremendously discouraged individual, the pessimistic response to the demands of the given real situation, in greater or lesser degree a lack of affirmation of life. This viewpoint alone, that of seeing the total individual in relationship to the totality of life, renders possible an understanding of the psychotic personality, of the meaning and significance of all

his different modes of expression. The psychotic is right; neither he nor this earth is capable of satisfying his exaggerated expectations. Lacking the courage to live a useful social life, he tries to rescue his self-esteem and to become a centre of attention by the useless method of a thoroughly discouraged individual. In this sense psychosis is egocentrism, maladjustment, a social disturbance, a deviation from the way of social tasks, a self-blockade. This holds true both in single and in periodic melancholia. The paranoic's goal of godlikeness is readily seen in his magnomania and persecution mania. The greatest contradiction of normal, social individuality is the schizophrenic personality. The schizophrenic is the most hypersensitive of all psychotics. The earth would have to be many times as pleasant as it is in order to be supportable to him. And so, depreciating it through his falsification of reality, his negativism, delusions, hallucinations, etc., he turns aside into a realm of his own creating. Onset, intensity, duration, frequency of relapses, improvement and healing of psychosis depend upon the strength of the inferiority feeling, the fixation of the goal, and the individual preparation for life in childhood. The difference between neurosis and psychosis is that in the former the pessimism, the inferiority feeling, the disheartenment are much less, and the sense of reality much greater, than in the latter. The neurotic makes a compromise with life, a half-half adjustment; he is social in so far as his superiority is approximately guaranteed. His goal of superiority is fictitious; whereas in psychosis, because of the much greater feeling of insecurity, the goal has become dogmatized and inexorable, an ideal that has to be taken without regard for the realities of life. The prognosis depends upon the degree of the patient's suffering from his psychotic method of living, and on his will to change it, i.e., on the strength of his inferiority feeling and the fixation of his goal. Treatment becomes a matter of explaining and encouraging, step by step, always according to the individual situation, i.e., of clearing up the inferiority feeling as an exaggeration, an understandable error, and the goal of godlikeness as unrealizable and too costly a sacrifice of the actual possibilities of life. More than all else, we must work to reinforce his sense of reality, his self-reliance his feeling of responsibility and his fellow-feelings.

C. S. R.

[59] **A study of 50 cases of psychopathic personality.**—G. E. PARTRIDGE. *Amer. Jour. Psychiat.*, 1928, vii, 953.

OF 21 males and 29 females studied, three main types were distinguished: delinquent type; inadequate type; and a general incompatibility type. There was a considerable variety among the incompatibles, but a fairly consistent general background of emotional instability and social ill-adjustment. A cursory survey of the histories makes clear that the aggregate of trouble which has centred about these 50 persons is great. The type of reaction which the patient may have adopted in early life is very persistent despite all ordinary efforts to change it. There is much psychopathy in the families of these

patients. In 24 cases psychopathic personalities in the direct line could be identified, or were strongly indicated. In seven more there was some form of psychopathy or neuropathy, and six of the other families were suspicious. Physical examination showed nothing noteworthy except that in 13 cases there were signs of involvement of endocrine glands, no judgment being possible, however, regarding the priority of the physical as contrasted with the personality disorder. Nothing outstanding appears from the enumeration of the occupational selection of these patients. In 21 of the cases there was recorded either attachment to one parent or antagonism. Twenty-two of the females and 10 of the males were married, or had been, but there was conspicuous lack of good marital adjustment. There was little evidence of overt homosexuality. The most conspicuous fact observed in the study was that in almost every case there was evidence of attitudes relating to feelings of insecurity or inferiority. The general conclusion is reached that the psychopath is one in whom strong demands are accompanied by feelings of inadequacy, inferiority, or insecurity, and in whom there developes a tendency to resort to one or more typical reaction patterns, different in different cases, but all to be included in a general way under the terms 'tantrums,' 'sulks,' and 'running away.'

C. S. R.

[60] **Blood chemical changes in the toxic psychoses.**—Howard D. McIntyre. *Amer. Jour. Psychiat.*, 1928, vii, 919.

Blood chemical examinations are of great value in the study of the early stages of mental disease, especially of those psychoses showing any symptoms of hallucinatory confusion. They assist in the differential diagnosis of the toxic psychoses, especially of those on a uræmic basis. They also aid in demonstrating the role of toxic factors in precipitating psychotic attacks in the major psychoses. Evidence is here presented to show that manic-depressive, schizoid and confusional reactions may in their early stages be accompanied by signs of toxæmia, viz. leucocytosis, acidosis, fever and nitrogen retention. Treatment directed towards the elimination of the toxæmia is frequently effective in bringing about a mental recovery. The toxins, acid or otherwise, so affect the hydrated colloids of the nerve cells as to bring about a cracking of the emulsion of which the nerve cell is composed. Treatment to be effective must take place before this irreversible cracking-point is reached.

C. S. R.

[61] **Long-section blood-sugar tolerance study in a case of depression.**—T. Raphael, W. C. Ferguson and O. M. Searle. *Arch. of Neurol. and Psychiat.*, 1928, xix, 120.

A case of manic-depressive psychosis, typical from all points of view, was studied from the standpoint of blood-sugar tolerance. It was found that in this disorder there is a tendency towards an abnormal reaction following the ingestion of glucose; this is shown by a curve of decreased tolerance resembling

that found in certain somatic disturbances, such as diabetes mellitus. This abnormality of carbohydrate metabolism appears to be phasic and to disappear with clinical recovery. It therefore seems that in this condition the patient is affected physiologically as well as psychologically, but whether the physiological factor is primary or secondary to the mental symptomatology is uncertain. In view of the diminished basal metabolic rate the disturbance in carbohydrate physiology probably represents merely a single manifestation of a general metabolic depression.

R. M. S.

[62] **The electrical skin resistance.**—C. P. RICHTER. *Arch. of Neurol. and Psychiat.*, 1928, xix, 488.

RICHTER measured the electrical skin resistance in twenty-six normal persons and in sixty-six psychopathic patients. The normal subjects gave records which remained fairly constant. In marked contrast, many of the patients presented records with large individual differences and at the same time great variation from the normal. These patients were all either catatonic or schizophrenic. The schizophrenic patients with marked feelings of passivity deviated to one extreme; those of the active or paranoid reaction type deviated to the other. The records from manic-depressive and hysterical patients deviated slightly, but were still within the limits of normal variation.

R. M. S.

PSYCHOPATHOLOGY.

[63] **Bacterial change in mental disorder: preliminary note on Morgan's bacillus.**—F. H. STEWART. *Jour. Ment. Sci.*, 1928, lxxiv, 269.

IF a tyrosine bouillon tube is inoculated with faeces from a healthy person, incubated for 48 hours and distilled, it will be found to contain phenol of not more than 0·008 per cent., or at the most 0·015 per cent. If, on the other hand, similar cultures are made from mental patients, they will contain 0·02 to 0·03 per cent. in half of the specimens examined. At least one-quarter of mental patients who are acutely ill carry B. Morgani: a smaller proportion carry B. phenologenes (Berthelot), and a still smaller proportion a phenol-producing paracolon. B. Morgani is the important phenol-producing bacterium of the insane. The strains of B. Morgani found in these cases do not cause diarrhœa, but the dead bacteria are toxic to man, causing marked local, and in some cases general reaction, when moderate doses are injected hypodermically. After intravenous injection of minute doses they cause pyrexia, and if the dose is larger or the kidneys already diseased, a transient nephritis.

As far as it is possible to judge by the few cases treated, efficient vaccination (hypodermic followed by intravenous) can remove B. Morgani from the bowel. Such removal is followed by improvement in bodily health and possibly improvement of the mental condition. C. S. R.

[64] **The erythrocyte sedimentation reaction: its value in psychiatry.**—J. GOLDWYN. *Arch. of Neurol. and Psychiat.*, 1928, xix, 110.

By erythrocyte sedimentation reaction is meant the speed with which the red blood cells settle in a column of citrated blood. The phenomenon has been found of definite clinical value in the field of general medicine, in surgical investigations and in obstetric and gynæcological studies. The author applied the reaction in different forms of mental disorder; his conclusions are as follows.

1. The acceleration of the erythrocyte sedimentation reaction varies directly with the amount of mental deterioration, the amount of organic destruction and the amount of toxicity present.

2. Unless the cases are complicated by physical disease, the sedimentation reactions are normal in cases of manic-depressive psychosis, psychopathic personality, psychoneurosis and paranoia.

3. Increased readings are found in all cases of senile psychosis, psychoses due to cerebral arteriosclerosis, general paralysis, neurosyphilis, psychoses showing mental deficiency or somatic disease, acute types of alcoholic psychoses and in many cases of epileptic psychosis and involutional melancholia.

4. In dementia præcox the simple and paranoid types tend to give normal readings, while the hebephrenic and chiefly the catatonic types tend to give slightly accelerated reactions. Cases showing marked mental deterioration give increased readings. No case of dementia præcox gave a marked increase unless complicated by some physical disorder.

5. The sedimentation reaction is of limited aid in psychiatric diagnosis.

6. Because the sedimentation test is a reliable and efficient indicator of many somatic diseases, it is of practical value in dealing with psychotic patients.

R. M. S.

[65] **Bacterial change in mental disorder: coliform group.**—F. H. STEWART. *Jour. of Ment. Sci.*, 1928, lxxiv, 58.

Paracolon or *mutabile* is to be found in 30 to 40 per cent. of mental patients, and in 17 per cent. of these patients the bacteria in question make up from 10 to 100 per cent. of the coliform flora. Carriers of paracolon-mutabile are at least four times more common among the insane than in the general community. Paracolon-mutabile occur in all forms of insanity, but more frequently and abundantly in acute illness than in chronic. In some recurrent cases which have been closely studied they are absent or few in number during intervals, increase rapidly at the onset of an attack, fluctuate with the symptoms, and disappear at convalescence; nevertheless in one and the same patient they may be present in large numbers in one attack and absent in the next.

The increase of these organisms is probably due to some change of environment in the bowel—a change which acts either by causing massive variation from colon to paracolon-mutabile, or by favouring overgrowth of small numbers

of the latter bacteria which may already be present. The change of environment postulated may take origin either in the secretions of the host or in those of some other intestinal micro-organism.

As to the practical question whether paracolon and mutabile take any part in producing mental disease, it is clear that the association which seems established between mental disease and increase of these organisms may be one of three alternatives: (1) a constitutional basis of mental disease may alter the secretions of the patient and so evoke the bacterial change. (2) The increase of paracolon-mutabile may be one of many causes capable of upsetting an unstable nervous system, and we have seen that paracolon-mutabile are actually more toxic than normal colon. (3) A substance, such, for example as phenol, if formed in the intestine may give rise to an increase of paracolon-mutabile, and after absorption cause the mental illness. Metchnikoff, in 1910, showed that phenol combined with sulphuric and glycuronic acid is lethal to guineapigs, and produces arteriosclerosis if given over long periods in sub-lethal doses. Wladytchko also found proliferation of neuroglia and neuronophagy in the brain of a guineapig which had been treated in this manner. It is possible that all three modes come into play in a series of vicious circles.

C. S. R.

[66] **Investigation of the permeability of the brain membranes in cases of mental disorder.**—HERBERT A. STRECKER. *Jour. of Ment. Sci.*, 1928, lxxiv, 73.

IN 22 cases of general paralysis the P.Q., i.e., the ratio of bromide found in the blood compared with that found in the cerebrospinal fluid, was below the normal average in 82 per cent. of the cases, although a few showed markedly decreased permeability. In 30 cases of dementia præcox the P.Q. was above the normal average in 36 per cent. of the cases, below in 30 per cent., the remainder being normal. N.A.B. treatment resulted in a definite increase of the permeability. The average P.Q. of five cases of delusional insanity and three cases of mania and manic-depressive insanity was normal. C. S. R.

[67] **On the pathology and laboratory diagnosis of paresis.**—F. PROESCHER and A. ARKUSH. *Jour. Nerv. Ment. Dis.*, 1928, lxvii, 21.

THE authors have found that iron pigment is not confined to the perivascular sheaths of the cortical vessels in general paralysis but may also be demonstrated in the adventitial cells of the cerebrospinal fluid. The method is as follows:

Centrifuge 5 c.cm. of cerebrospinal fluid. To the sediment add 5 c.cm. of the following solution:—

96 per cent. alcohol, 95 c.cm.;

aqueous solution of ammonium hydro-sulphide, 5 c.cm.

Centrifuge again and examine deposit. Iron is indicated by the presence of deep-black granules in the adventitial cells. Black particles elsewhere are without diagnostic significance. The reaction appears to be specific for general paralysis. R. M. S.

[68] **Spirochaetosis of the central nervous system in general paralysis.**—Robert R. Dieterle. *Amer. Jour. Psychiat.*, 1928, vii, 547.

A case of juvenile paralysis is described as an example of Jahnel's vascular type of spirochætosis. The vast number of organisms and their general topographical distribution, their presence in the principal regions of the cortex, in the basal ganglia, and in the cerebellum, is an unusual instance of such intense and widely disseminated occurrence. The brain seems to be the most favourable medium for the growth of the spirochæta pallida. The localization of the swarms within the cortex at the level of the middle zone is in direct relation to the complex capillary network existing between the long and short branches of the cortical blood-vessels. The absence of organisms within the white substance of the convolutions, the presence of great masses of them in the middle zone of the cortex and the smaller subpial colonies, suggest that the lymph carries some organisms outward towards the arachnoid. The metastasis of spirochætes undoubtedly occurs lymphogenously in a local sense, and hæmatogenously in a wider sense. Cases of intense spirochætosis are found when death occurs during a period of mental confusion, the remission indicating a period of lessened organismal activity. Thus there exists a correlation between clinical course and spirochætosis. It is believed that physical factors and the mechanical effects induced thereby have an influence upon the perivascular arrangements of the spirochætes, in addition to the lymphophilic factors. The histopathological alterations of the basal ganglia in direct relation to the spirochætosis lead one to believe that the primary invasion by the organisms is into this region. The spirochæta pallida is in relation to both vascular systems, whereas the virus of lethargic encephalitis most commonly affects the ganglionic division alone. The choroid plexuses were negative for spirochætes.

C. S. R.

[69] **Observations on changes in demeanour as a whole consecutive to brain lesions** (Beobachtungen über die Veränderungen des Gesamtverhaltens bei Gehirnschädigung).—Kurt Goldstein. *Monats. f. Psychiat. u. Neurol.*, 1928, lxviii, 271.

The author remarks with justice on the disadvantages of the prevailing tendency to attempt to associate limited defect of function with specific lesion in cases of brain injury or disease. Such "general" symptoms as have been noted in these cases are usually considered to concern memory, attention, volition, and so forth. But alterations in the personality as a whole, in general demeanour and conduct, have received less notice, although from time to time they have been made the subject of investigation. In this communication the author gives the results of his study on the attitude adopted by the sufferer towards his own defects, and on his insight into his own condition. It is pointed out how frequently he is unaware of his infirmity in the first place, and

how frequently he is capable of greater degree of function that might be expected from isolated examination of the particular defect concerned. One explanation of the latter circumstance is that a certain restoration of balance as between individual and environment takes place; the organism that is restricted in function by disease or injury develops an altered relation to environment. Such changes as would threaten the continuance of important functions are involuntarily ignored; the patient "rises superior" to his blindness, deafness, and so on, rendering his milieu more adequate to his restricted activities.

A careful description is also given of certain peculiarities in the conduct of these patients—their tendency to seek quiet, their orderliness, their discomfort in the absence of concrete stimuli, their avoidance of empty space. This last feature is of no little interest and is well exemplified in the clinical illustrations supplied.

S. A. K. W.

[70] **The acute psychiatric type of epidemic encephalitis.**—Irving J. Sands. *Amer. Jour. Psychiat.*, 1928, vii, 975.

Epidemic encephalitis may first manifest itself in an acute psychotic reaction. This may be either a delirium or of an occupational nature, or may consist of episodic outbreaks of silly excitement, impulsive acts, delusional or bewildered states, confusion and marked fear. The subsequent appearance of neurological symptoms helps the observer to arrive at the correct diagnosis. A review of the literature and illustrative cases are presented.

C. S. R.

[71] **Encephalitis lethargica and the interpretation of mental disease.**—Ives Hendrick. *Amer. Jour. Psychiat.*, 1928, vii, 989.

Study of the literature and of fifteen cases at the Boston Psychopathic Hospital has revealed no unequivocal evidence of a fundamental relationship between the symptoms and the patient's personality prior to the encephalitis, although the clinical picture may be superficially coloured by his individuality and environmental experience. The antisocial behaviour and emotional instability of the encephalitic seem to be chiefly the result of a pathological defect in the ability to control impulses which are common to the race, rather than the product of the disease and a specially conditioned personality. Many symptoms found in encephalitic psychoses resemble closely those in autochthonous disorders, especially schizophrenia; but the parallelism of the fundamental features of such diseases appears to be exceptional. Parkinsonism and impairment of general intelligence are common, in adults as well as in children with psychotic features. The patient's consciousness of mental and physical inferiority, and his responsiveness to enlightened discipline, have been too little emphasized.

C. S. R.

[72] **Peduncular hallucinosis** (L'hallucinose pédonculaire).—L. VAN BOGAERT. *Revue neurol.*, 1927, i, 608.

THE author's case is that of a woman with evidence of a left hemiplegia, which under treatment gradually improved, but other symptoms were present, such as an ataxic gait, tendency to falling backwards, adiadochokinesis, nystagmus; in addition, hypomanic attacks occurred in the evenings, with visual hallucinations. The details of the case were furnished at greater length in an earlier communication (*Revue neurol.*, April, 1924).

At autopsy was found an area of softening involving the regio subthalamica, cerebral peduncle, nucleus ruber, and extending to the anterior corpora quadrigemina and the pulvinar of the thalamus, in a forward direction, and to the upper part of the pons posteriorly. The author attributes the whole symptomatology of his case to the discovered peduncular lesion. In his own words: "Here is a syndrome of visual hallucinosis and of evening hypomania occurring with a lesion strictly peduncular, and the practically complete integrity of the cortex and subcortical nuclei has in this case the greatest significance." He proceeds to an interesting if speculative discussion on the possibility of mental symptoms of extracortical origin, basing his deductions on his interpretation of the case.

The reviewer feels that these deductions raise in an acute form the question of principles of localisation, and of attribution of symptoms solely to recognisable structural lesions. In his opinion it is a mistaken conception which sets down positive symptoms to negative lesions and ignores the possibility of alteration of function in mechanisms whose anatomical basis is not the seat of obvious structural change.

S. A. K. W

[73] **Mental disorder in cardiac disease.**—CAREY F. COOMBS. *Jour. of Ment. Sci.*, 1928, lxxiv, 250.

THE author concludes that there is no evidence that cardiac disease can, unaided, cause insanity. There is reason to believe that mental disturbances can cause cardiac disorder, but whether these can go further and establish organic change appears uncertain. Many morbid processes affect both heart and brain, and thus account for some of the coincidences between cardiac disease and insanity. C. S. R.

[74] **Pellagrous insanity.**—T. C. COOPER. *Amer. Jour. Psychiat.*, 1928, vii 945.

THE relationship which various nervous and mental disturbances bear to pellagra has not as yet been definitely settled. Only 4 to 10 per cent. of pellagrins are said to develop a psychosis and it is often impossible to determine whether the pellagra is the primary condition and the psychosis secondary or the reverse. The majority probably belong to the latter. There is no symptom-complex that can be said to be pathognomonic of pellagrous insanity.

Probably the first and most persistent mental symptom is that of a mild depression combined with a certain amount of apathy. A pellagrous mania has been described, but neither of these conditions is characteristic of the manic-depressive psychosis. In the early stages the depression is mostly apathy; later there is an amentia, with loss of memory, confusion, hallucinations and paraesthesias, from which there arise morbid impulses and delusions. Following, there develops a true dementia, and at least in some cases there is an increased pressure of the spinal fluid and an œdema of the brain. In the acute cases of the so-called typhoid type there is usually fever and delirium with hallucinations of sight and hearing. This is ascribed to a toxic state which is often secondary to some known infection. In the majority there is some anæmia with general asthenia. There is also some degeneration of the spinal cord, especially in the posterior and lateral columns, and to a lesser extent the anterior columns. This causes some uncertainty of gait, a spasticity of the muscles, and various paræsthesias, and there is a hyperactivity of the deep reflexes. There is no specific treatment. Personal hygiene and a diet rich in vitamins, especially vitamin B, with plenty of rest and sleep, are the essentials. Moderate doses of some of the arsenicals are helpful. In some cases where there is marked confusion and agitation it has been found that a spinal puncture with a removal of 15 to 30 cubic centimetres of fluid has given some relief for a time, but this should be considered as a measure that is still in the experimental stage.

C. S. R.

[75] **Emotional factors in nutrition work with children.**—GEORGE J. MOHR. *Mental Hygiene*, 1928, xii, 366.

PHYSIOLOGICAL processes concerned with nutrition bear a distinct relationship to the emotional state of the individual and are modifiable by emotional changes. The earliest situation is dependent upon an emotional relationship between mother and child. Disturbances here may lead to immediate difficulty in providing proper nutriment for the child. Properly utilized, however, this situation offers a powerful means for the establishment of routine and the formation of habits that may simplify the question of feeding. Exaggeration of this situation may lead to the development on the part of the child of behaviour designed to prolong the infantile state of dependence. In children who present feeding problems, if we rule out all cases in which there is disease, or organic or functional incapacity of some sort, there is still a residue of cases with unexplained symptoms. In these, neurotic manifestations may be recognised, and here the emotional element is of primary importance. The child may be utilising a neurotic device to attain an understandable end or the mother may induce the difficulties by anxieties and fears; more frequently the situation is a rather subtle one in which there is much emotional interplay between the various members of the family who ordinarily have close contact

with the child. Such emotional-nutritional situations may be prevented, but considerable insight on the part of the mother is required. Temporarily, complete separation of the child from the family group may be necessary and is usually quickly effective in developing in the child more useful modes of behaviour. C. S. R.

PROGNOSIS AND TREATMENT.

[76] **Therapeutic mechanism of malaria treatment of general paralysis: observations on a case.**—Goeckeman. *Amer. Jour. Med. Sci.*, 1928, clxxv, 261.

The author records a case which was diagnosed clinically and serologically as general paralysis. There was no evidence of previous cutaneous lesion. The woman was treated with malaria, but had no further antispecific treatment after this. Five months later she developed typical nodulo-ulcerative syphilides on the right buttock and left thigh.

He points out that it has been generally held that neurosyphilides are evidence of an allergic condition of the tissues and indicate partial immunity. Cases which show them run a benign course if they develop neurosyphilis later. He suggests that the appearance of a syphilide in his case, five months after malaria treatment, points to the conclusion that malaria treatment helps to re-establish an immunity in which the skin takes an important part. The gradual improvement in the spinal fluid also supports the conception that malaria produces a tissue reaction rather than a direct spirochæticidal effect.

P. W.

[77] **Note on the treatment of general paralysis by apyrexial malaria.**—F. H. Stewart. *Jour. of Ment. Sci.*, 1928, lxxiv, 276.

In the course of experimental work, it was decided to try the effect of two short courses of malaria, stopping the attacks by quinine in doses of 30 gr. daily as soon as the first rigor as was convenient.

The advantages claimed for this method of treatment are (1) reduction of mortality and of suffering for the patient; (2) mental and bodily re-education can be begun a few days after the first attack; (3) antisyphilitic treatment by tryparsamide or salvarsan can be combined with the malarial treatment more readily; (4) great reduction in anxiety and labour for the physician and nursing staff; (5) the treatment is placed within the reach of the general practitioner since shielded nursing in hospital is no longer necessary.

C. S. R.

[78] **Two years' experience with the malarial treatment of general paralysis in a State institution. Clinical, serological, and autopsy observations in 100 cases.**—Max A. Bahr and W. L. Breutsch. *Amer. Jour. Psychiat.*, 1928, vii, 715.

Of 100 paralytic patients, treated with malaria, 25 were able to leave the hospital. Twelve patients improved to such a degree that they can be trusted

in a useful occupation in the hospital. Forty patients are placed in the unimproved group. In 32 patients of the latter group the disease is progressing. In 8 instances the disease has remained stationary. Five patients succumbed during the rigors and 18 died following the malaria.

Serological changes following malarial treatment occurred in both the improved and unimproved groups. While there was no parallelism between the degree of clinical and serological improvement, the percentage of the serological improvement, particularly as far as the colloidal gold curve was concerned, was higher in the improved groups. The paretic gold curve was reduced in intensity but remained of the paretic type. The conversion of a paretic curve into a true luetic one in these cases was an exception.

It cannot be denied that this form of treatment is attended with definite risk for the patient. With a better knowledge of the complications that arise during the rigors, deaths due to therapeutic malaria will be reduced.

C. S. R.

[79] **Influence of malarial treatment on the spinal fluid in general paralysis.**—H. A. BUNKER. *Arch. of Neurol. and Psychiat.*, 1928, xix, 478.

OBSERVATIONS on the spinal fluid of patients treated by induced malaria appear to be of prognostic significance only in a negative sense. A satisfactory response is not necessarily of favourable import; but the few cases in which reduction of the cell count does not take place with more or less promptitude or in which pleocytosis reappears, and those in which the total protein in the spinal fluid remains at or returns to a level in excess of 100 mg. per 100 c.cm., seem to offer definite evidence of a minimum therapeutic result. The behaviour of the Wassermann reaction in the spinal fluid does not seem to possess a decisive significance; but whether the cases with a more negative reaction will compare favourably with those with a less negative one regarding the ultimate duration of the therapeutic result achieved is a question to be answered in the future.

R. M. S.

[80] **Treatment of mental diseases with African tick fever** (Quelques essais thérapeutiques de maladies mentales par la fièvre récurrente africaine). —D'HOLLANDER and DE GREEFF. *Jour. de Neur. et de Psych.*, 1927, xxvii, 555.

CASES of melancolia and of dementia præcox in its incipient and established stages, and one case of general paralysis were treated with a mild form of African tick fever. The results were nil, but the interest of the communication lies in the fact that the infection of Dutton's spirochæte was obtained from a culture.

J. P. M.

[81] **Contributions to the psychotherapy of organic diseases** (Beiträge zur Psychotherapie bei Organerkrankungen).—STAUDACHER. *Münch. med. Woch.*, 1926, 2020.

IT is suggested that the field of psychotherapy may usefully be widened, instead of letting quacks deal with cases thought unsuitable—but really capable of benefit by such treatment.

The author, whose method is suggestion under hypnosis, describes fully two cases which he treated when they thrust themselves upon him after he had refused them as being wholly organic and not suitable, but which proved to have a large functional element and to derive great benefit from psychotherapy.

The first was that of a 50-year-old inn-keeper, who had had a corneal wound in 1917 during the war, and had been under constant treatment for pannicular conjunctivitis since 1926. He sought psychotherapy for headaches, giddiness and sleeplessness, which the author at first refused, thinking them attributable to the eye condition. However, his whole state improved so much under suggestion that this was also applied to the eye symptoms, and resulted in a great lessening of the inflammation, so that after four weeks the patient could bear light without his dark glasses, and could leave off the daily eye drops, which he had previously failed to be able to do, in spite of repeated trials. This improvement was still maintained three months later without further treatment.

The second was a case of chronic nephritis, also of nine years' duration, following on acute trench nephritis. The patient had albuminuria, had recently had uræmic convulsions, and albuminuric retinitis. He complained of severe headaches, general weakness, and impaired vision. The fear of blindness played a large role in a general anxiety state, and he became generally nervous, depressed and sleepless. The convulsions were of a mixed type with a considerable hysterical element in some of them.

Here again, treatment was undertaken unwillingly by the author, at the request of the patient and his doctor and relatives, and the results were surprisingly beneficial; from being hardly able to walk he regained a fair degree of activity, sleep and headaches and vision improved, and with the general improvement and hopeful mental attitude even the albuminuria was reduced considerably. In the after-history of this case there were several relapses, aborted by prompt suggestion under hypnosis.

The author suggests that the mechanism of such improvement is probably the influence of the emotions acting through the vegetative nervous system, bringing about changes of vascularity which affect the course of a chronic organic condition; a hopeful outlook and increased desire for health thus promote natural healing tendencies. In cases where there is a considerable emotional factor and neurotic elements superadded he urges that doctors should include psychotherapy in their treatment. M. R. B.

[82] **Prisons as laboratories for personality study.**—WILLIAM A. WHITE. *Mental Hygiene Bull.*, 1928, vi, 5.

SOCIETY has made its pronouncement as to what sort of activities are undesirable from its point of view, but the problem of crime will never be solved as long as we fail to make an accurate survey of the personality and problems of the antisocial offender. There was a time in the history of crime and penology when inanimate objects and animals were tried and executed. Inanimate objects and animals have been set apart from the ritual of punishment, and even children have come to be regarded as outside of that domain of the law. Nowadays children, the obviously defective, and the obviously insane are no longer punished by the criminal law, but their defect is largely in the intellectual field. We are willing to exclude from the ritual of punishment and put in appropriate educational establishments or institutions for social guidance people whose intellectual level makes them irresponsible. But we are not prepared as yet to deal in the same way with people the emotional aspect of whose life is equally undeveloped. Persons who have an intellectual age of five or six years are no longer considered responsible, but those who have an emotional level of five or six years are still considered responsible.

In order to deal with the problem of crime we must understand the criminal in all of his various aspects—the social, the intellectual, the affective, the economic, and others. Added to that, we need a revaluation of the fundamental concepts of the criminal law, such as punishment, guilt, responsibility, and a revaluation of what crime itself really means.

C. S. R.

Reviews and Notices of Books.

Die Labyrinthreflexe auf die Augenmuskeln nach einseitiger Labyrinthexstirpation. By Dr. R. LORENTE DE NÓ, Assistant in the Cajal Institute, Madrid. With 186 figures in the text, and 22 plates. Berlin and Vienna: Urban and Schwarzenberg. 1928. Pp. 205. Price Mk. 15.

WHEN a closely printed book of over 200 pages is seemingly required for a discussion of a part only of the functions of the labyrinth some idea of the intricacy and complexity of labyrinthine activity may be gained, but these are no more in evidence than is the uncertainty still surrounding it. Dr. de Nó appears almost overwhelmed by the amount of ignorance still obtaining in regard even to the elements of the subject. He cites with cordial approval the dictum of Gudden: "Erst Anatomie und dann Physiologie, wenn aber zuerst Physiologie, dann nicht ohne Anatomie," and then proceeds to show, as might be expected of a member of the Cajal school, how grossly incomplete even anatomical knowledge of labyrinthine central structures and connexions is. He gives beautiful drawings of vestibular nuclei and emphasises the fact that we do not know what three-quarters of them represent in anatomical relationships. "Es ist traurig, aber es ist wirklich so."

The major part of the work is devoted to an account of the results of personal experiments on the neural mechanisms underlying the action of the labyrinth on ocular reflexes, and it leads to a highly technical investigation of the function of the semicircular canals along mathematical lines. In a concluding chapter a précis is given of the author's conceptions, together with a critical review of some current theories The technical nature of the book makes it rather hard reading even for those who are abreast of current research, but Dr. de Nó does not wish it otherwise. He repeatedly stresses his point, that hitherto schematisation of labyrinthine functions with a view to simplicity has proved a serious obstacle in the path of real advance.

Incidentally, the neurologist for whom perhaps some of this specialised investigation has but a secondary interest ought to note with particularity certain conclusions drawn in respect of mesencephalic function and the place occupied by the red nucleus in decerebrate states. Dr. de Nó has proved convincingly that longitudinal sections in medulla and pons, and transverse sections in the latter also, can and do produce decerebrate rigidity, as well as disappearance of existing righting-reflexes. In all of these animals the red nucleus was preserved intact, whence the conclusion is, that "the nucleus ruber cannot be the centre for the origination of righting-reflexes, or for the normal

apportionment of tone in the extremities." He therefore cannot support the contentions of Magnus and Rademaker; on the contrary, his experiments suggest that the centres are in the substantia reticularis.

S. A. K. W.

Neurobiologische Studien am menschlichen Foetus. By M. MINKOWSKI.

Methoden der Reflexprüfung bei Menschen. By ARTHUR BÖHME. Section No. 253 of the Handbuch der biologischen Arbeitsmethoden, edited by Geh. Med.-Rat Prof. Dr. EMIL ABDERHALDEN. Berlin and Vienna: Urban and Schwarzenberg. 1928. Pp. 511-592. Price Mk. 10.

THE first contribution in this section of Prof. Abderhalden's monumental Handbuch deals with the physiology of the fœtus and opens up a new field. As can be understood, the living material, derived almost entirely from Cæsarean sections, has a very brief survival period and must be utilised quickly and systematically, Full details of the author's technique in this respect are furnished. It is not a little surprising, perhaps, that he has been able to investigate not merely the comparatively simple matter of reflexes (cutaneous and deep) but also the postural reactions of Magnus and de Kleijn and the electrical reactions of muscles; further, by appropriate experimentation attempts have been made to determine function at different neural levels.

Following Jacksonian conceptions, Prof. Minkowski draws an analogy between the results of dissolution effected by disease and the types of functional activity found in the fœtus, but he states in more than one place that this likeness must not be taken too strictly.

In the second contribution the reader is given a very complete account of the reflexes, usual and unusual, employed in clinical medicine, embracing not a few which deserve greater attention than is commonly allotted them. Of value are the references to the original descriptions. The term "reflex" is used with considerable latitude, since phenomena are here included which belong rather to the groups of spinal automatisms and mesencephalic reactions. The sole question in this respect that does not appear to be adequately dealt with is that of cortical "reflexes," and this is the more regrettable since they are now realised to be of considerable significance.

Die Psychocathartische Behandlung nervöser Storüngen. By Dr. LUDWIG FRANK. Leipzig: Georg Thieme. 1927. Pp. 208.

THE author adopts the term "thymopathy" for the psychoneuroses in general and apparently also for some psychoses, and advocates a method of treatment which he believes to be efficacious in them. The essence of the method is the induction of a state of "half-sleep"—a state between sleeping and waking in which active attention is abolished, and when "unconscious" trends come to consciousness in the form of visual images, accompanied by their appropriate

emotion. This process he designates as a kind of abreaction, and the appearance in consciousness of the images is assumed to be a matter of free association. There is, therefore, nothing at all new in the method except its restriction to the half-waking state. He describes its application in a number of conditions, in a very general way.

R. D. G.

Die Grundlagen der Psychoanalyse. By Dr. HEINZ HARTMANN. Leipzig: Georg Thieme. 1927. Pp. 192. Price M. 9.60.

THE author is concerned to display psychoanalysis as a science and its methods as scientific methods. He points out that its concepts and bearing are biological, and he relates certain psychoanalytic findings to results obtained in the laboratories of experimental psychology. There are chapters, among others, on free association, on the unconscious, on psychical dynamics, on symbolism, instinct, the libido-theory, and on the relation of psychoanalysis to psychiatry. They contain convenient summary expositions of the basic Freudian assumptions, with a defence of the latter where a defence seemed necessary. The chapter on the relations of psychoanalysis and psychiatry emphasises the importance of psychoanalytic contributions to the study of the content in the psychoses, and claims priority for psychoanalysis in the systematic investigation of the personality. The psychoanalytic interpretation of the real significance of precipitating factors is also elaborated. There is a commendable caution in the author's attitude towards the would-be psychoanalytic explanation of essential epilepsy. The book is a useful summary and defence of psychoanalytic theory, on a broad basis.

R. D. G.

Stammering. A Psychoanalytic Interpretation. By ISADOR H. CORIAT, M.D. (Nervous and Mental Disease Monograph Series, No. 47.) Nervous and Mental Disease Publishing Co., New York and Washington. 1928. Pp. 68.

THIS monograph is an amplification of contributions on the subject previously published by the author in various journals. Different theories of stammering—cerebral congestion, transient auditory amnesia, spastic neurosis of speech, localized motor obsessional neurosis, hereditary tic—are briefly considered and found to be unsatisfactory from many points of view. In the following chapters the writer's views are elaborated at some length to show that stammering is a psychoneurosis and that its rational treatment and prevention must be on psychoanalytical lines. Stammering is regarded as a regression to the earliest level of childhood, at which stage in this respect there is an arrest of development. The individual has remained fixed at the oral stage of libido, finds morbid gratification in his symptom by means of which he tenaciously retains his earliest source of pleasure, that of nursing. There is also a conflict produced by ego resistance against betrayal through speech of certain thoughts,

mainly sexual in nature. Evidence is brought forward to support these views and the stammerer is stated to have character traits which confirm this interpretation.

The author says that the conception here given is not a mere theoretical speculation or a philosophical system of ideas, but is the result of practical experience in psychoanalysis. Considering the inadequacy of current methods of attacking stammering, this psychoanalytical viewpoint should receive due recognition, whether or no all its details can be accepted. No psychopathologist can afford to neglect the study of the conceptions treated of in these pages. There is, however, much repetition, which might have been avoided with advantage. A bibliography is appended. C. S. R.

The Struggles of Male Adolescence. By C. STANFORD READ, M.D. London: Fisher Unwin. 1928. Pp. 243. Price 7s. 6d.

THIS is a clearly written book on the problems confronting the growing youth. It is unusually just in the weight which it apportions to the various factors that constitute his difficulties, and introduces Freudian interpretations in reasonable proportions and in a common-sense way. The book is divided into two parts, the first dealing with the emotional forces at work in the course of development, the second with the consequences of conflict between them and the methods of resolving the conflict. In the chapter on heredity stress is properly laid on the importance of environmental factors in moulding the individual, and this leads to a hopeful attitude towards the treatment of the major and minor symptoms that are succinctly and clearly described in the later part of the book. Other chapters deal with the sexual instinct, and the emotional influences of home and of school. The discussion of corporal punishment is a moderate and enlightened one. The chapter on parental problems contains some wise remarks. On the whole the book tends to over-emphasise the importance of sexual difficulties, but not in any far-fetched way, and it should be helpful to parents and teachers.

Clinique Médicale des Enfants: Affections du Système Nerveux. By P. NOBÉCOURT, physician to the Hôpital des Enfants-Malades, Paris. With 70 illustrations. Paris: Masson et Cie. 1928. Pp. 374. Price 45 fr.

IN a series of clinical lectures delivered at various times during the last few years and now published in book form, Professor Nobécourt gives a lucid and interesting demonstration of the clinical aspects of such familiar conditions as epidemic encephalitis, Sydenham's chorea, tuberculous meningitis, meningococcal meningitis, pneumoccocal meningitis, poliomyelitis, diphtheritic palsy, facial palsy, and Friedreich's disease; other chapters are devoted to headache in childhood, to the nervous complications of typhoid fever, to congenital syphilis, sleepy states, and herpes zoster. In a pleasantly readable fashion he furnishes us with clinical histories, sketches in outline his methods of diagnosis, and duly emphasises his difficulties and hesitations when the case is not clear. Etiology and treatment are dealt with at some length,

Taken as a whole, the volume exemplifies the clinical method at its best and can be commended to neurologist and pediatrist alike.

The Examination of the Central Nervous System. By DONALD CORE, M.D. (Manc.), F.R.C.P. (Lond.). Edinburgh: E. & S. Livingstone. 1928. Pp. 239. Price 8s. 6d. net.

THIS little book can be commended as a useful guide for the junior student. It is intended as a purely clinical handbook, but even so its value would have been enhanced had more space been allotted to simple anatomical and physiological data. Ordinary routine methods of examination are well described and there are useful hints on the taking of case-histories and the general diagnosis of nervous diseases. The illustrations are not, however, particularly good.

Malattia di Parkinson e Sindromi Parkinsoniane. By Prof. Dr FEDELE NEGRO, lecturer in neuropathology at the University of Turin. Turin: S. Lattes and Co. 1928. Pp. 295.

DR. FEDELE NEGRO has republished in a greatly augmented form the monograph on Parkinson's disease which we reviewed favourably on its original appearance in 1923 (see this JOURNAL, Feb. 1924, p. 381). It deals in a critical fashion with the numerous still unsettled problems connected with the condition, and may be consulted with advantage by all who are interested in these. Unfortunately, it contains neither table of contents nor index. The bibliography is helpful.

Travaux et Mémoires. Tome deuxième. By PIERRE MARIE. Paris: Masson et Cie. 1928. Pp. 390. Price 30 francs.

WE have already noticed in these columns the first volume of this re-issue of the works of Professor Pierre Marie. The present volume contains articles on the Charcot-Marie-Tooth type of muscular atrophy, on lacunar forms of hemiplegia and on hereditary cerebellar ataxia, as well as a large number of other and slighter papers. All the articles are full of interest to the neurologist, who will be glad to add these volumes to his bookshelves as a worthy monument to the great clinical acumen of their distinguished author.

Tonic Hardening of the Colon. By T. STACEY WILSON, M.D., B.Sc. (Edin.), F.R.C.P. (Lond.) Humphrey Milford, Oxford University Press. 1927. Pp. 210.

THE author of this book is evidently an enthusiast on his own subject and as such can hardly expect his readers to follow him entirely in all his views. An interesting account is here given of what the author claims is an exceedingly common condition, with full descriptions of its symptomatology and treatment. A very wide scope is claimed for this disorder as a causal factor in many conditions, including many functional disorders of the nervous system. The collection of aphorisms with which the book concludes sufficiently indicates the variety of states in which the author maintains that tonic hardening of the colon may play a part. The reader will probably be wise in exercising discrimination in accepting all these claims.

Occasional Notes.

A REPORT ON THE THIRD GERMAN MEDICAL CONGRESS FOR PSYCHOTHERAPY, BADEN-BADEN, 1928.

By GEORGE GERÖ, Vienna.

In Germany the interest in psychotherapy is continually increasing. Not only specialists who deal with the psychically suffering but also the general practitioner, the gynæcologist, the internist and the tuberculosis expert wish for enlightenment with regard to the problems and the technique of this new science and art. This desire can readily be understood, the fundamental issue of the psychotherapeutic movement being the idea that it is always man as an entity who is the object of the whole activity and thought of the physician, and that disease is frequently a compound of connected physical and psychical factors. That is why efforts are being made to elaborate, consciously and systematically, methods of psychical treatment which have always been intuitively used by the best physicians, and to elevate them to the rank of an important part of the general equipment of the modern medical man.

At this meeting, which was the third of its kind, many general practitioners were present who were looking out for information with regard to the results and the development of psychotherapy, and evidently found it. As a matter of course the participation of the specialists has been equally active, the aim of these meetings being a combined one. Primarily they act as an intermediary in delivering to the general practitioner a notion of the general problems of psychotherapy, but on the other hand (the dissension between the different schools representing strictly opposite opinions having become a rather radical one) they try to clear the way for a mutual understanding.

This time the two schools struggling for supremacy were the individual psychology of Dr. Alfred Adler and Freud's theory of psychoanalysis. Individual psychology was represented by the reports of Dr. Seif (Munich), Dr. Wexberg (Vienna), and Dr. Künkel (Berlin). Their psychoanalytical opponents were Dr. Schultz-Hencke (Berlin) and Professor Schilder (Vienna).

Dr. Seif gave an outline of the point of view of individual psychology with regard to the problem of neurosis. According to his explanations the neurosis is a social disturbance of interhuman relations, an excuse and an effort to justify the discouraged and egocentric individual in escaping from his life-work. Accordingly, the cure consists in educating the discouraged and dependent individual to become courageous and independent by converting his egocentric attitude into an appropriate one which makes him fit for life.

Dr. Wexberg reported on organ inferiority in connexion with certain results of internal medicine. According to his specifications Adler's doctrine of organ inferiority represents the extension of the principle of the point of minor resistance to the sphere of clinical pathology. Whilst the doctrine of constitution assumes partly the conception of a specific disposition for certain diseases, partly that of a constitutional debility of the whole organism, Adler asserts a predisposition of certain organs and organic systems for diseases of a different kind. There are families, for instance, with dispositional debility of the stomach-intestine system, which manifests itself in one case by an ulcus ventriculi, in another by gastric carcinoma, in a third case, it may be, by a dyspepsia. Dr. Wexberg is of opinion that this connexion is nowadays often accepted in constitutional research as well as in practical internal medicine. Less attention is paid to another section of Adler's doctrine, the compensation of organ inferiority. This super-compensation does not change an inferior organ into a normal one, but into an "emphasized" one. The psychologically important consequence of the compensation of an organ inferiority shows itself also in the fact that in the end the entire organism seems to be grouped round the "emphasized organ" as its centre.

Dr. Schultz-Hencke, one of the psychoanalytic speakers, declared all the theses of individual psychology to be implicitly contained in psychoanalysis, while the reverse is not the case. Individual psychology only inquires in what direction the symptoms are tending, whereas psychoanalysis also searches for the causes. He does not deny that individual psychology is performing satisfactory service in the range of what psychoanalysis calls "the secondary profit of disease," that is, demonstration of the actual psychical situation which favours the fixation of symptoms. But its range is far narrower, considering the enormous extension of the field, than that of psychoanalysis.

Professor Schilder analyzed Dr. Wexberg's report and gave a fundamental criticism of Alder's doctrine of organic inferiority. He pointed out that Adler's doctrine is, using a term of the modern psychology of form (*Gestaltpsychologie*), a typical piece-work (*stückhaft*). The inferior organ has no isolated place in the whole of the organism. How it may manifest itself depends on temperament, on strength of the affects and on the intellect of the entire personality. The problem of constitution reaches far too deep to be capable of being unlocked by the formula of organ inferiority.

The second subject of discussion was the problem of character. What is character, and how is it to be grasped? Professor Haeberlin (Basle), Dr. Allers (Vienna), and Dr. Klages (Zürich) tried to investigate these intricate problems.

Professor Haeberlin pointed out the tasks set for scientific characterology, and the means by which it is to solve them. The principal work of characterology is to constitute a psychologically and logically ordered system of the points in which two individual characters may differ. Characterological differences must follow the connexion of actions. This connexion is determined first by its form, and secondly by its content. Such formal characterological standpoints are, for instance, constancy of character, its differentiation, course of action, intelligence, memory, and so forth. The content is the interest manifesting itself through actions.

Dr. Allers, also, asserted that understanding of character must be based on the analysis of its actions. Every action tends towards a definite goal which always consists in establishing an entity. Besides, the action is in any case the settling of a tension out of which it has arisen, and which is cancelled through it. According to Dr. Allers character cannot be understood from the structure of impulses, nor does he acknowledge its being determined by innate moments of constitution. He defines character as the formal principle of the total of all reactions of a human being. Since action, being the architype of reaction, is directed towards values, character may be defined as the substance of individual laws of value-preference.

The third subject of the Congress was the relation between experimental psychology and psychotherapy. The lecturer, Professor Lewin (Berlin), pointed out in an extremely interesting paper that experimental psychology nowadays has become capable of treating successfully problems of emotional and impulsive life, i.e., of spheres of essential importance in therapy. It is by no means an essential quality of the experiment to keep apart from real life. In psychotherapeutic treatment everything depends on grasping the individual peculiarities of the case. In experimental psychology nowadays less stress is laid on the static procedure of research of laws, based on the experimental penetration of the dynamic structure of a relatively few concrete cases. Whether the experiment keeps nearer to or farther from real life does not depend upon its successful imitation of definite situations of life in its intensity, but on its establishing processes of action and the structure of systems, dynamically equivalent to the cases of everyday life in question. It is even possible to arrange situations within the field of laboratory-experiments wherein the person investigated is not aware of his being part of the experiment—experiments exempt from task. Dr. Lewin described experiments trying to explain tension and detension of need-systems, the degree of communication between different needs and psychical spheres, the question of repression, the different kinds of surrogate gratification, experiments which are already contributing by their essential results to throw light on these processes.

A series of lectures completed the programme. The lively debates after the lectures proved the active interest taken in these questions by the medical world.